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RESPIRATORY CARE

A MONTHLY SCIENCE JOURNAL
38TH YEAR—ESTABLISHED 1956

■ Editorials

Walking the Walk—Not Just Talking the Talk: Cigarette Smoking and RCPs

Adventures in Recycling: The Reuse of "Disposable" Pulse Oximeter Probes

MEDWATCH: The New FDA Medical Products Reporting Program

■ Preliminary Evaluation of High-Frequency Chest Compression for Secretion Clearance in Mechanically Ventilated Patients

■ Cyanosis and Sulfhemoglobinemia: A Case Report

■ Noninvasive Bi-Level Positive Pressure Ventilation: Management of Two Pediatric Patients

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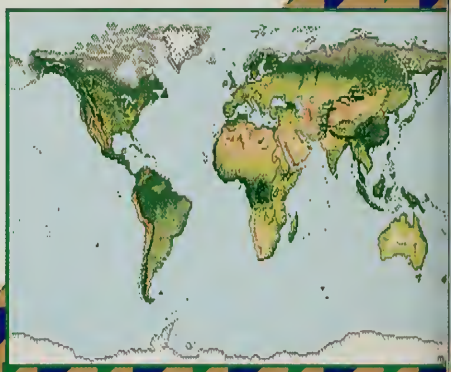

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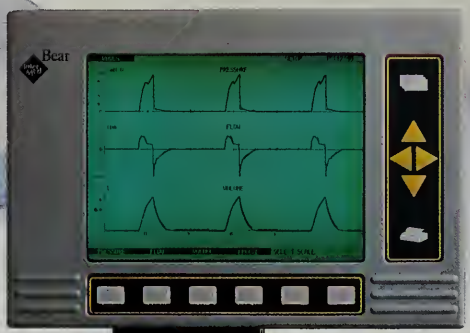

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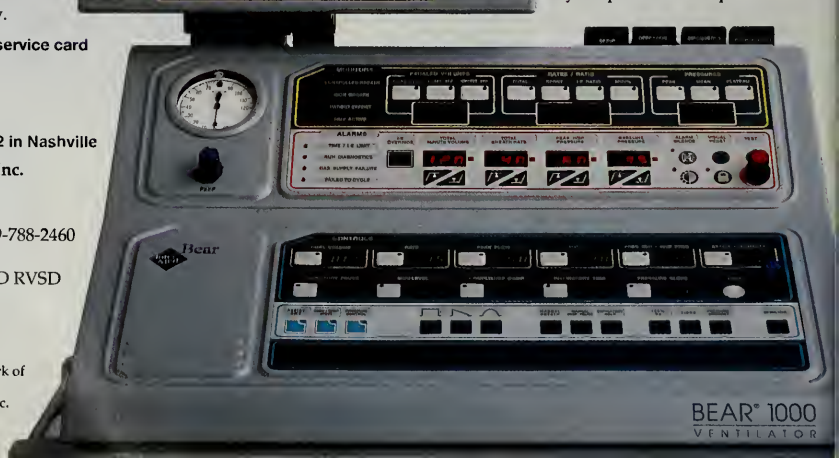

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CONTENTS

October 1993
Volume 38, Number 10

EDITORIALS

1069 Walking the Walk—Not Just Talking the Talk: Cigarette Smoking and Respiratory Care Practitioners
by Yvonne Jo Robbins, Douglas C Albright, and E Thomas Deutsch Jr—Bryn Mawr and Malvern, Pennsylvania

1072 Adventures in Recycling: The Reuse of "Disposable" Pulse Oximeter Probes
by John W Salyer, Karen Burton, Joanna Lynch, Julie Ballard, and Jim Kennan—Salt Lake City, Utah

1077 MEDWATCH: The New FDA Medical Products Reporting Program
by Dianne L Kennedy--Rockville, Maryland

ORIGINAL CONTRIBUTIONS

1081 Preliminary Evaluation of High-Frequency Chest Compression for Secretion Clearance in Mechanically Ventilated Patients
by Judy Whitman, Ron Van Beusekom, Suzanne Olson, Mary Worm, and Frank Indihar—St Paul, Minnesota

CASE REPORTS

1088 Cyanosis and Sulfhemoglobinemia: A Case Report
by James M O'Connor—Lancaster, Pennsylvania

1092 Noninvasive Bi-Level Positive Pressure Ventilation: Management of Two Pediatric Patients
by Olugbenga A Akingbola, Gina M Servant, Joseph R Custer, and John M Palmisano—Ann Arbor, Michigan

CLASSIC REPRINTS

1099 RETROSPECTROSCOPE REDUX: How To Make Hasenpfeffer
by Julius H Comroe Jr (reprinted, with permission, from the American Review of Respiratory Disease 1975;111:849-852)

TEST YOUR RADIOLOGIC SKILL

1103 Arrest following a Prolonged Course of Periodic Coughing and Fever in a Child
by Lynn M (Rasmussen) Sullivan and Robert M Kacmarek—Boston, Massachusetts

BLOOD GAS CORNER

1107 Blood Gas Corner #31—A Patient Presenting with Leg Pain and Shortness of Breath
by Joseph E Ollivier—Fresno, California

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CONTENTS, *Continued*

October 1993
Volume 38, Number 10

BOOKS, FILMS, TAPES, & SOFTWARE

1110 Guidelines for Pulmonary Rehabilitation Programs, by the American Association for Cardiovascular and Pulmonary Rehabilitation (AACVPR), edited by Gerilynn Connors BS RCP RRT and Lana Hilling RCP, authored by Committee Members C William Bell PhD MBA, Howard M Kravetz MD, Kathleen Morris RN MS RRT, and Andrew L Ries MD
reviewed by David P Meeker and Kay Stelmach—Cleveland, Ohio

1110 Respiratory Care: A Guide to Clinical Practice, 3rd edition, edited by George E Burton MD, John E Hodgkin MD, and Jeffrey J Ward MEd RRT
reviewed by Vasudevan C Jaidev—Charlottesville, Virginia

LETTERS

1112 Deja Vu! A CPR Device to the Attention of MEDWATCH
by Robert R Fluck Jr—Syracuse, New York

1113 Objections to Humidification Editorial
by Daniel I Sessler—San Francisco, California; with response by Richard D Branson and Robert L Chatburn—Cincinnati and Cleveland, Ohio

1117 Reply to Paluch on HME Misrepresentation
by Geoff Lloyd and Kenneth Graf—Portsmouth, England, and Glen Cove, New York

1119 Deep Breaths, Sighs, and Pulmonary Artery Catheters
by Ted Reesor—Burlington, Ontario, Canada

ABSTRACTS

1050 Summaries of Pertinent Articles from Other Journals

NOTICES

1123 Examination Dates, Notices, Prizes

CALENDAR OF EVENTS

1121 Meeting Dates, Locations, Themes

INDEXES

1124 Authors in This Issue
1124 Advertisers in This Issue
1066 Advertiser Help Lines

Editorials, Commentaries, and Reviews To Note

Bronchoalveolar Lavage in Asthma: An Update and Perspective (state-of-the-art review)—DL Smith, RD Deshazo. Am Rev Respir Dis 1993;148:523.

MDI Workshop: Standards for In Vitro/In Vivo Measurements (conference summary)—International Society for Aerosols in Medicine: North American Chapter, McMaster University. J Aerosol Med 1993;6:123.

Pressure-Preset Ventilation—Part I: Physiologic and Mechanical Considerations (review)—PB Blanch, M Jones, AJ Layon, N Camner. Chest 1993;104:590.

The Laryngeal Mask Airway: Its Uses in Anesthesiology (review)—JH Pennant, PF White. Anesthesiology 1993; 79:144.

Clarifying the Role of Continuous Fick Cardiac Output Measurement during Exercise (editorial)—PD Lumb. Chest 1993;104:332. (Pertains to Nakanishi et al paper abstracted on Page 1057.)

Childhood Tuberculosis: A Diagnostic Dilemma (editorial)—JR Starke. Chest 1993;104:329. (Pertains to Delacourt et al paper abstracted on Page 1060.)

Profound Hypercarbia Late in the Course of Laparoscopic Cholecystectomy: Detection by Continuous Capnometry (case report)—D Hall, A Goldstein, E Tynan, L Braunstein. Anesthesiology 1993;79:173.

Tracheal Perforation: A Complication associated with Transtracheal Oxygen Therapy (case report)—AS Menon, BW Carlin, PD Kaplan. Chest 1993;104:636.

Paradoxical Bronchoconstriction with Nebulized Albuterol but Not with Terbutaline (case report)—JP Finnerty, PH Howarth. Am Rev Respir Dis 1993;148:512.

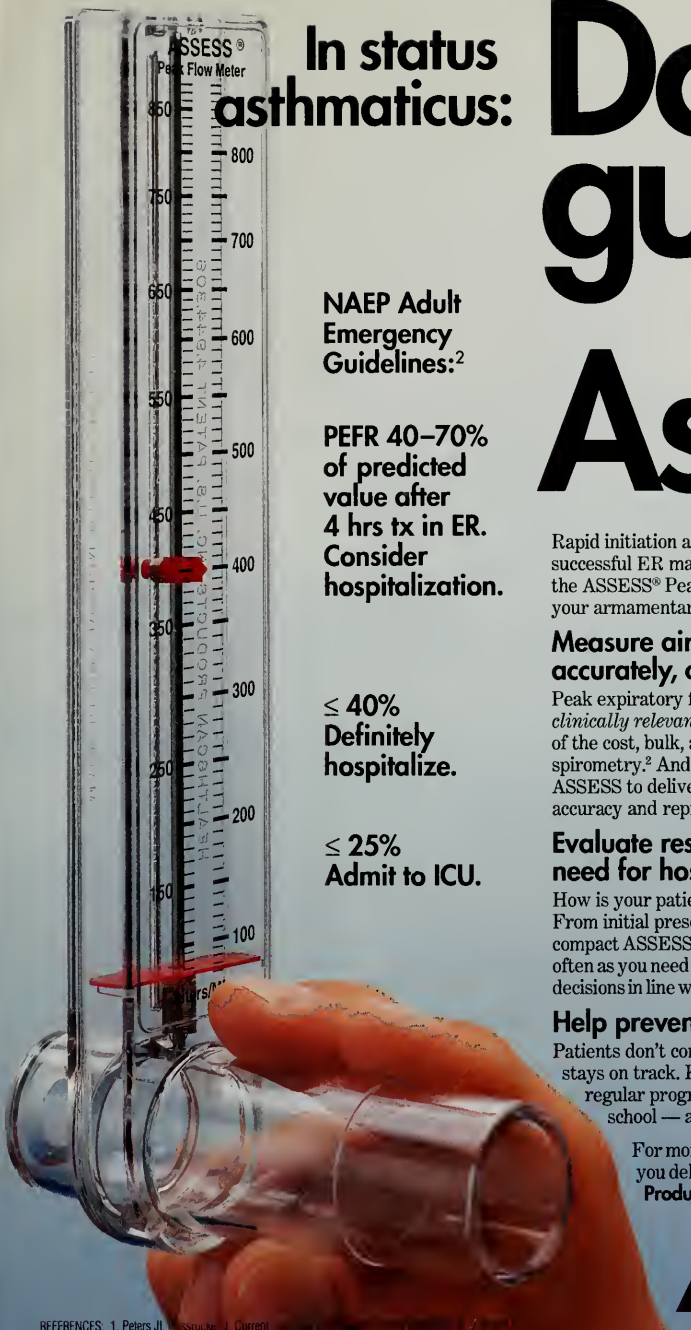
Role of Pulse Oximetry in the ICU (editorial)—SJ Barker. Chest 1993;104:330. (Pertains to Inman et al paper abstracted on Page 1063.)

Spirometry in the Asthmatic Child: Is FEF_{25-75} a More Sensitive Test Than FEV_1/FVC?—P Lebecque, P Kiakulanda, AL Coates. Pediatr Pulmonol 1993;16:19.

The use of spirometry in the assessment ot children with asthma is taking on new importance with the realization that considerable airway obstruction may exist in the absence of clinically detectable abnormalities. There has been controversy over the value and relative sensitivity of various spirometric indices. This study evaluated the forced expiratory flow between 25% and 75% of vital capacity (FEF_{25-75}), forced expired volume in 1 second (FEV_1), and the ratio between the FEV_1 and the forced vital capacity (FVC) in 100 asthmatic children aged 6-17 years, 29 of whom were wheezing at the time of the evaluation. All children with clinical wheezing had a $FEF_{25-75} < 2$ standard deviations (SD) below the mean (−2 SD), whereas 8 had a normal FEV_1. The majority of the wheezing children had abnormalities of all 3 indices, whether expressed as −2 SD or, in the case of the FEV_1/FVC, arbitrarily taken as < 80%. Sixty-seven children of the entire study group had at least 1 abnormal spirometric index, but 38 of these had no clinical abnormalities. Twelve children had a reduced FEF_{25-75} as the only abnormality. These results suggest that FEF_{25-75} is a sensitive index of airway obstruction.

Mediators of Septic Shock: New Approaches for Interrupting the Endogenous Inflammatory Cascade—BP Giroir. Crit Care Med 1993;21:780.

OBJECTIVES: To review the molecular pathogenesis of septic shock, with particular emphasis on the induction of cytokines by endotoxin.



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By understanding the mechanisms that result in the systemic inflammatory response, novel clinical interventions may be more effectively studied. DATA SOURCES: The English medical literature was reviewed, including human clinical trials, animal experiments, and *in vitro* studies elucidating cellular and molecular interactions. Expert testimony from the Roundtable Conference on Sepsis (Brussels, March 1992) was also used to synthesize emerging concepts and to ensure inclusion of ongoing investigations. STUDY SELECTION: Emphasis on controlled experimental studies which elucidated the molecular and cellular interactions during sepsis. DATA EXTRACTION: This study focused only on data that directly involved the induction and regulation of protein mediators of sepsis, especially tumor necrosis factor (TNF) and interleukin-1. Data concerning the role of TNF during health were extracted from the author's peer-reviewed data. DATA SYNTHESIS: Information concerning the many facets of the systemic inflammatory response was integrated into a chronological, clinically oriented model of cytokine induction during endotoxemia. CONCLUSIONS: The induction of inflammation during sepsis is a complex, but increasingly understood, biological cascade that is dependent on inter- and intracellular signaling. Novel biotherapies may improve patient outcome in sepsis by interrupting any or all points of signal transduction.

Increased Physiological Dead Space during Exercise after Bronchodilation in Cystic Fibrosis—G Kusenbach, F Friedrichs, H Skopnik, G Heimann. Pediatr Pulmonol 1993; 15:273.

Bicycle ergospirometry was performed on 14 patients with cystic fibrosis (CF), for evaluating the effects of salbutamol and theophylline on the ventilatory response to exercise. After 1 week without bronchodilator therapy the patients cycled at one-third and two-thirds of their individual maximal working capacity (Wmax). The test was repeated three times after treatment with salbutamol, theophylline, or both drugs, respectively. After the combined therapy, physiological deadspace, ventilation, ventilatory equivalent of oxygen, and end-expiratory oxygen pressure increased significantly during steady state exercise at one-third Wmax. Similar, although not statistically significant changes, were observed after monotherapy with salbutamol or theophylline and during exercise at two-thirds Wmax. These effects could not be predicted by any lung function tests at rest or by the Shwachman-Kulczycki score. The results indicate that in some patients with CF bronchodilators can impair lung function during exercise. In conclusion, the effects of medication on exercise performance of patients with CF have to be considered. Especially, the use of bronchodilators requires a careful evaluation of their real benefit in each individual patient.

Dexamethasone Treatment Suppresses Collagen Synthesis in Infants with Bronchopulmonary Dysplasia—E Co, G Chari, K McCulloch, D Vidyasagar. Pediatr Pulmonol 1993;16:36.

Collagen is an essential component of connective tissue and is present in the pulmonary interstitium. Collagen deposition is known to increase in many acquired chronic diseases, including bronchopulmonary dysplasia (BPD). Urinary excretion of hydroxyproline has been used as a specific index of collagen synthesis. Many studies have demonstrated that dexamethasone therapy is associated with respiratory improvement in infants with BPD but the mechanism of this effect is not well understood. We postulated that in infants with BPD who receive dexamethasone, suppression of collagen synthesis may cause respiratory improvement. Therefore, we studied the effect of dexamethasone on respiratory status and urinary excretion of hydroxyproline in 14 ventilator-dependent infants with BPD. Infants received 0.5 mg/kg/day dexamethasone, tapered by half every 3 days to complete a 12 day course. Eleven of the 14 infants were extubated at a mean ± SD of 8.7 ± 4.9 days after starting dexamethasone. Mean urinary hydroxyproline/creatinine ratios at 3, 6, 9, and 12 days of dexamethasone therapy were significantly lower than the mean pretreatment value, but after discontinuation rapidly rose toward baseline values. Decreased urinary excretion of hydroxyproline indicates that dexamethasone suppressed collagen synthesis in these infants. We speculate that suppression of collagen synthesis reduced pulmonary inflammation and fibrosis, resulting in respiratory improvement.

Gender Related Differences in Airway Tone in Children—LI Landau, W Morgan, KS McCoy, LM Taussig. Pediatr Pulmonol 1993;16:31.

The effects of gender, volume history, and inhaled atropine and isoproterenol on lung mechanics were assessed in 16 normal boys and 14 normal girls using lung volumes, flow-volume curves, and oscillatory resistances. Flows were measured from full and partial forced expiratory flow-volume curves. Six girls and 6 boys were studied before and after inhaled atropine, and 10 boys and 8 girls before and after inhaled isoproterenol. Girls demonstrated a significant increase in flows on full and partial curves with a deep inspiration [$\dot{V}$max-partial 0.73 ± 0.34 (SD) to $\dot{V}$max-full 0.80 ± 0.37 and

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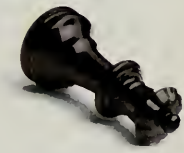

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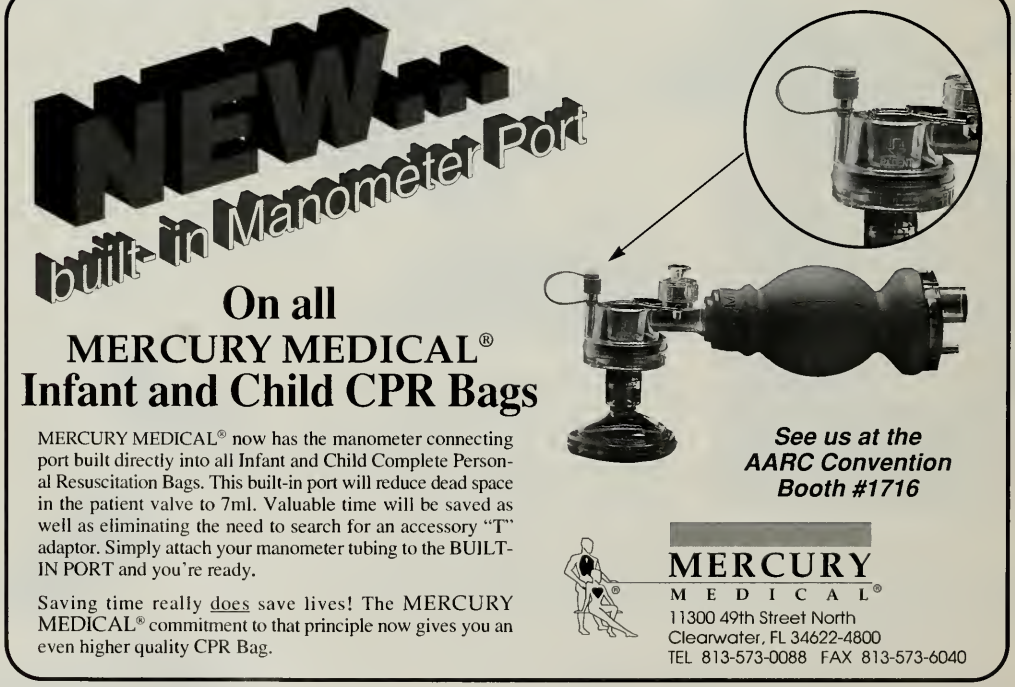
0.83 ± 0.20 to 1.06 ± 0.29 TLC/s in each group] and following inhalation of isoproterenol on the partial curves only (0.73 ± 0.34 to 0.93 ± 0.40 TLC/s). Boys showed a small but significant increase in $\dot{V}$max with isoproterenol on full curves but not on partial curves. Following atropine, boys demonstrated a significant increase in $\dot{V}$max on partial flow-volume curves (0.78 ± 0.28 to 1.00 ± 0.35 TLC/s) and a significant decrease in specific respiratory resistance (7.6 ± 2.7 to 5.1 ± 0.9 cm $H_2O \cdot s \cdot L^{-2}$), whereas girls had no such changes. These data suggest that boys have greater resting airway tone than girls and that this tone is less responsive to deep inspiration and isoproterenol independently, although a combination of isoproterenol and a deep inspiration will produce increased flows in boys. Atropine reduces airway tone predominantly in boys, suggesting that the increased resting airway tone in boys is partially mediated via the vagus nerve.

Examination Gloves as Barriers to Hand Contamination in Clinical Practice—RJ Olsen, P Lynch, MB Coyle, J Cummings, T Bokete, WE Stamm. JAMA 1993;270:350.

OBJECTIVE: To test the effectiveness of vinyl and latex gloves as barriers to hand contamination with gram-negative organisms and enterococci during routine hospital procedures. DESIGN & INTERVENTIONS: We studied 137 procedures during which a health care worker's gloved hand contacted a patient's mucous membrane and was thus potentially contaminated with gram-negative rods or enterococci. Quantitative hand cultures were obtained from each health care worker before and after the gloved contact using a modified glove juice method, and the exterior glove surface was also quantitatively cultured after patient contact. Used gloves were then tested for leaks using the American Society for Testing and Materials' watertight test. SETTING: Harborview Medical Center, a 330-bed city-county hospital and level 1 regional trauma and burn center, is both a teaching facility affiliated with the University of Washington and the major provider of care to indigent and uninsured persons in Seattle-King County, Washington. PATIENTS & OTHER PARTICIPANTS: Respiratory therapists performing endotracheal tube care on intubated intensive care unit patients, registered nurses performing digital rectal stimulation for bowel training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on patients with spinal cord injury in the rehabilitation ward, and dentists performing routine dental examinations and procedures on healthy outpatients in the dental clinic. MAIN OUTCOME MEASURE & RESULTS: Eighty-six of the 135 gloves cultured had gram-negative rods or enterococci on the external surface after use and were thus sources of potential hand contamination. Microbial contamination of the health care worker's hands occurred in 11 (13%; 95% confidence interval, 6% to 20%) of these 86 events, and was more frequent with vinyl (10 of 42) than latex (one of 44) gloves ($p < 0.01$). After use, glove leaks were also more frequent in vinyl gloves (26 of 61) than with latex gloves (six of 70) ($p < 0.001$). Even when leaks were present, gloves prevented hand contamination in 77% of instances and quantitative counts of microorganisms contaminating hands were 2 to 4 logs less than counts on external glove surfaces. Health care workers reported awareness of the presence of glove leaks in only seven (22%) of the 32 events in which leaks were subsequently demonstrated. CONCLUSIONS: Under conditions of routine use, gloves effectively function as a protective barrier even when leaks are present. Latex gloves were less frequently associated with leaks and hand contamination. Since hand contamination occurred after 13% of exposures and cannot be readily identified by health care workers, routine hand washing should be done after each patient contact.

Out-of-Hospital Cardiac Arrest: Racial Differences in Outcome in Seattle—MR Cowie, CE Fahrenbruch, LA Cobb, AP Hallstrom. Am J Public Health 1993;83:955.

OBJECTIVES: Out-of-hospital sudden cardiac arrest is a key area in which to study the dual problem of the poorer health status of minority populations and their poorer access to the health care system. We proposed to examine the relationship between race (Black/White) and survival. METHODS: We determined the incidence and outcome of cardiac arrests in Seattle for which medical assistance was requested. RESULTS: Over a 26-month period, the age-adjusted incidence of out-of-hospital cardiac arrest was twice as great in Blacks than in Whites (3.4 v 1.6 per 1,000 aged 20 and over). The initial resuscitation rate was markedly poorer in the Black victims (17.1% v 40.7%), and rates of survival to hospital discharge were also lower in Blacks (9.4% v 17.1%). Both effective initial resuscitation and survival were significantly related to White race following adjustment for other covariates. CONCLUSION: The differences in outcomes were not fully explained by features of the collapse or relevant service factors. Possible explanations include delays in instituting therapy, less bystander-initiated cardiopulmonary resuscitation, poorer levels of health, and differences in the underlying cardiac disorders.

Work-Site Smoking Policies: Their Population Impact in Washington State—S Kinne, AR Kristal, E White, J Hunt. Am J Public Health 1993;83:1031.

This article presents data from a population-based, random-digit dialing telephone survey of 1,228 employed adults in Washington State, conducted 1989 through 1990. Eighty-one percent of men and 91% of women reported work-site smoking restrictions. Employees in work sites with no-smoking policies were less likely to be current smokers; men in work sites with policies restricting smoking smoked fewer cigarettes on both workdays and nonworkdays. Forty-eight percent of male and 53% of female smokers reported reduced smoking as a result of a work-site policy. Work-site smoking policies, intended to protect against smoke exposure, may also reduce employee smoking.

Randomised Double-Blind Controlled Trial of Effect of Morphine on Catecholamine Concentrations in Ventilated Pre-Term Babies—MW Quinn, J Wild, HG Dean, R Hartley, JA Rushforth, JWL Puntis, MI Levene. Lancet 1993;342:324.

A sick premature baby who requires intensive care will undergo many uncomfortable procedures. It is now accepted that such babies perceive pain and need adequate analgesia, but little is known about the effects of sedation in these patients. We investigated the use of morphine to provide analgesia and sedation for ventilated pre-term babies in a randomised, double-blind, placebo-controlled trial. 41 mechanically ventilated babies who had been treated with surfactant (Curosurf) for hyaline membrane disease were randomly assigned morphine in 5% dextrose (100 μg/kg per h for 2 h followed by 25 μg/kg per h continuous infusion) or 5% dextrose (placebo). Plasma catecholamine concentrations were measured 1 h after the first dose of surfactant and 24 h later. Blood pressure was measured at study entry and after 6 h. The morphine and placebo groups showed no differences in method of delivery, Apgar scores, birthweight, gestation, or catecholamine concentrations at baseline. Morphine-treated babies showed a significant reduction in adrenaline concentrations during the first 24 h (median change –0.4 [95% CI –1.1 to –0.3] nmol/L, $p < 0.001$), which was not seen in the placebo group (median change 0.2 [–0.6 to 0.6] nmol/L, $p = 0.79$). There was a nonsignificant reduction in noradrenaline concentration in the morphine group. Blood pressure showed a slight but nonsignificant fall (me-

dian –4 mm Hg) in morphine-treated babies. The incidence of intraventricular haemorrhage, patent ductus arteriosus, and pneumothorax, the number of ventilator days, and the numbers of deaths did not differ significantly between the groups. Morphine, in the dose regimen we used, is safe and effective in reducing adrenaline concentrations in pre-term ventilated babies.

Management of Obstructive Sleep Apnea Syndrome in the Home: The Role of Portable Sleep Apnea Recording—MP Coppola, M Lawee. Chest 1993;104:19.

Unattended four-channel sleep apnea recording has been shown to be an accurate tool in the diagnosis of moderate to severe obstructive sleep apnea. We selected 11 patients with severe obstructive sleep apnea who had an apnea-hypopnea index (AHI) determined by unattended sleep apnea recording. The mean AHI was 41 (SD, 17.5). We began nasal continuous positive airway pressure (NCPAP) at home empirically with 5 cm to 7.5 cm of pressure for several nights. We then adjusted the level of NCPAP after telephone interview with the patients and their significant others. The level of NCPAP was increased by 2.5-cm increments until the patients reported cessation of snoring and symptom improvement. The mean NCPAP was 8.0 cm (SD, 1.4). We repeated the overnight sleep apnea recording while on NCPAP in all patients at home to determine their response to therapy. All 11 patients had documented return of their AHI to normal (mean AHI, 2.4; SD, 1.6). Statistically significant improvement was noted in the number of obstructive apneas, hypopneas, total respiratory events, and the AHI. Follow-up data confirmed that patients had improvement in their symptoms and remained compliant with therapy (mean follow-up = 18 months; SD, 10.2). No serious complications were encountered when NCPAP was introduced in an unattended setting. We were able to diagnose and treat these patients in an entirely outpatient setting.

Evidence for Transmission of *Pseudomonas cepacia* by Social Contact in Cystic Fibrosis—JRW Govan, PH Brown, J Maddison, CJ Doherty, JW Nelson, M Dodd, AP Greening, AK Webb. Lancet 1993;342:15.

Pulmonary colonisation with *Pseudomonas cepacia* in patients with cystic fibrosis can be associated with increased morbidity and mortality. The modes of transmission of *P cepacia* are, however, unclear. We used selective media and phenotypic and genomic typing systems to investigate the acquisition of *P cepacia* by adults with cystic fibrosis. An analysis of isolates from 210 patients attending regional clinics in Edinburgh and Manchester between 1986 and 1992 showed that the main cause of increased isolations of *P cepacia* from 1989 was the emergence of an epidemic strain that had spread between patients in both clinics. Epidemiological evidence indicated that social contact was important in spread of the epidemic strain within and between clinics. We suggest that guidelines to limit the acquisition of *P cepacia* should not be restricted to patients in hospital, and that intimate or frequent social contact is associated with a high risk of cross-infection.

Hemodynamic Effects of Oxygen Therapy in Patients with Acute Exacerbations of Chronic Obstructive Pulmonary Disease—A Esteban, E Cerda, MA De La Cal, JA Lorente. Chest 1993;104:471.

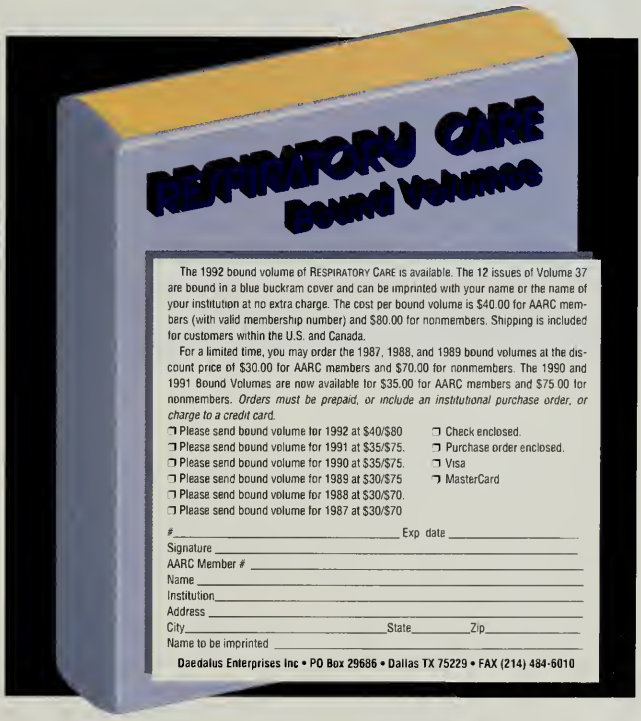
The effects of oxygen therapy in patients with stable COPD have been previously reported; however, the hemodynamic changes induced by oxygen therapy in patients during acute exacerbations of COPD are less well known. To investigate the hemodynamic effects of controlled oxygen therapy in patients with acute exacerbations of COPD shortly after arriving at the hospital, we studied 15 consecutive patients who came to the emergency room with acutely decompensated COPD that did not require mechanical ventilation. Patients were monitored with a pulmonary artery catheter and a radial artery catheter. Oxygen uptake was calculated by the modified Fick equation. Arterial and venous blood gas levels and hemodynamic parameters were measured while breathing room air (baseline) and after 30 min on oxygen therapy via face mask. Measurements were repeated after 24 and 48 h. The fractional concentration of oxygen in the inspired gas (F_{IO_2}) administered was adjusted to keep the P_{aO_2} above 55 mm Hg. All patients had a P_{aO_2} below 45 mm Hg at the beginning of the study. After 30 min of oxygen therapy, there was a significant ($p < 0.05$) increase in arterial oxygen saturation (from 62 ± 16 to 87 ± 9%), mixed-venous oxygen pressure (from 25 ± 5 to 43 ± 11 mm Hg), and oxygen delivery (from 11.1 ± 3.7 to 19.3 ± 8.9 mL · kg^{-1} · min^{-1}). Oxygen uptake did not change significantly (from 4.1 ± 1.2 to 4.3 ± 1.6 mL · kg^{-1} · min^{-1}). The oxygen extraction ratio decreased from 37.5 ± 10.1 to 25.3 ± 9.6%. These changes were maintained during the following 48 h. There were no significant changes in cardiac output and systemic vascular resistance. A trend toward lower values of pulmonary vascular resistance did not reach statistical significance. We conclude that oxygen therapy in patients with acute exacerbations of COPD that do not require mechanical ventilation increases oxygen delivery without changes in cardiac output or oxygen uptake.

Continuous Fick Cardiac Output Measurement during Exercise by Monitoring of Mixed Venous Oxygen Saturation and Oxygen Uptake—N Nakanishi, T Yoshioka, Y Okano, T Nishimura. Chest 1993; 104:419.

To determine whether continuous Fick cardiac output measurement is applicable to exercise testing, cardiac output data obtained by the continuous Fick method (Q_{cf}) during exercise were compared with data obtained by the thermodilution method (Q_{th}). Seventeen patients with old myocardial infarction underwent a 1-min or 3-min incremental exercise test (Protocols 1 and 2, respectively). During exercise, the oxygen consumption ($\dot{V}_{O_2}$), arterial oxygen saturation (S_{aO_2}), and mixed venous oxygen saturation ($S_{\bar{v}O_2}$) were monitored continuously. Q_{cf} was calculated at 12-s intervals by the Fick equation. The S_{aO_2} remained almost constant during exercise. The $S_{\bar{v}O_2}$ showed four characteristic phases during exercise Protocol 1. $S_{\bar{v}O_2}$ values changed rapidly in Phases 2 and 4, but only slightly during Phase 3. In exercise Protocol 2, $S_{\bar{v}O_2}$ almost reached a steady state by the end of each stage. The correlation between Q_{cf} and Q_{th} was good in Protocol 1 ($r = 0.86$), except in Phases 2 and 4, and was also good in Protocol 2 ($r = 0.80$). We conclude that the continuous Fick method may be applicable for determining the cardiac output during exercise provided that the variation in $S_{\bar{v}O_2}$ is slight.

Intraoperative Transesophageal Echocardiography for the Detection of Cardiac Preload Changes Induced by Transfusion and Phlebotomy in Pediatric Patients—DL Reich, SN Konstadt, M Nejat, HP Abrams, J Bucek. Anesthesiology 1993;79:10.

Intraoperative blood volume changes are difficult to monitor in pediatric patients. The authors tested the hypothesis that transesophageal echocardiography would identify changes in cardiac filling resulting from manipulations of blood volume. METHODS: Eleven patients (3-15 kg) were studied following sternal closure after repair of congenital heart lesions. Transesophageal echocardiography of the midpapillary left ventricular short axis view and hemodynamics were recorded at baseline (T1) during withdrawal of blood until the systolic blood pressure decreased by 5 mm Hg (T2) and 10 mm Hg (T3) and after reinfusion of the blood (T4). The identical cycle of blood withdrawal and reinfusion was repeated after administration of calcium chloride (10 mg/kg; T5-T8). RESULTS: Manually traced transesophageal echocardiography images of the left ventricular end-diastolic area decreased from 4.64 ± 1.50 cm^2 at T1 to 4.03 ± 1.25 cm^2 at T2 to 3.78 ± 1.35 cm^2 at T3, and increased to 4.42 ± 1.75 cm^2 at T4. Nearly identical results were obtained at T5-T8. End-systolic areas significantly decreased from 1.96 ± 0.86 cm^2 at T1 to 1.52 ± 0.73 cm^2 at T2 to 1.41 ± 0.62 cm^2 at T3, and increased to 1.87 ± 0.88 cm^2 at T4. An experienced anesthesiologist-echocardiographer blinded to study events was able to identify mild reductions in blood volume (T2, T3, T6, T7) from recorded cineloop video recordings with high sensitivity (80-95%) and specificity (80%). CONCLUSIONS: Transesophageal echocardiography is a potentially useful monitor of cardiac filling changes in pediatric patients.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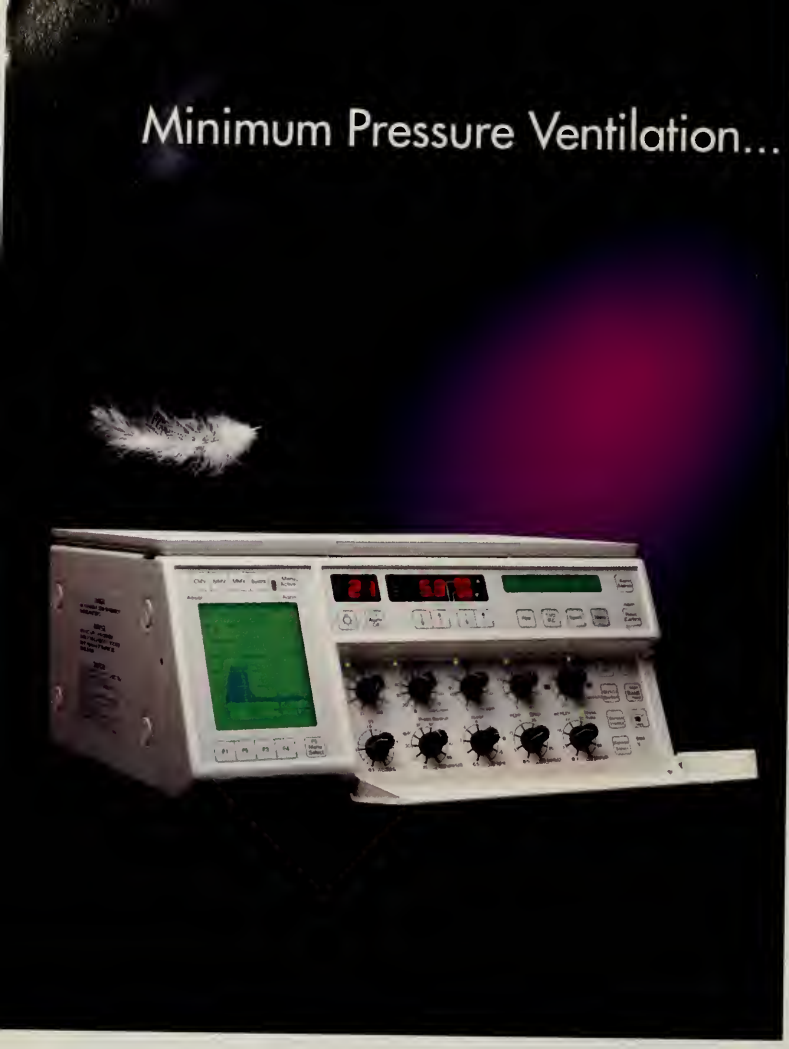

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Race and Birthweight in Biracial Infants—JW Collins Jr, RJ David. Am J Public Health 1993;83:1125.

OBJECTIVES: The purpose of the study was to determine the role of infant race as a determinant of the Black-White disparity in low birthweight (< 2,500 g). METHODS: Univariate analysis and multivariate logistic regression were performed on Illinois vital records from 1982 and 1983 and on 1980 United States census income data. RESULTS: Fourteen percent of the infants born to Black mothers and White fathers were of low birthweight, compared with 9% of infants born to White mothers and Black fathers and 6% of a random sample of White infants. Both groups of biracial infants were more likely to have been born to unmarried mothers and to reside in very low-income (< $10,000 per year) census tracts than were White infants. When all confounding variables were entered into a logistic model, the adjusted odds ratio of low birthweight for biracial infants born to Black mothers and White fathers equaled 1.4. When biracial infants born to White mothers and Black fathers were compared with White infants, the adjusted odds ratio of low birthweight equaled 1.0. CONCLUSIONS: Paternal and consequent infant race does not affect the birthweight distribution of those born to White mothers and Black fathers. Unidentified factors closely related to maternal race underlie the Black-White disparity in infant birthweight.

Increased Frequency of Obstructive Airway Abnormalities with Long-Term Tracheostomy—JH Law, K Barnhart, W Rowlett, O de la Rocha, S Lowenberg. Chest 1993;104:136.

Eighty-one patients with long-term tracheostomy tubes (mean duration, 4.9 months) were examined via fiberoptic bronchoscopy prior to decannulation. Obstructive airway lesions were observed in 54 patients (67%). All tracheal lesions were anatomically located proximal to the stoma. No cuff lesions were observed. The two most commonly observed lesions were tracheal granuloma (60%) and tracheomalacia (29%). Less frequently observed lesions were tracheostenosis (14%) and vocal cord and laryngeal dysfunction (8%). As a result of the high frequency of tracheal abnormalities, especially that for tracheal granuloma which has not been previously reported (to our knowledge), we recommend that all decannulation candidates undergo anatomic examination of the airways.

A Randomized Study of Outpatient Treatment with Ceftriaxone for Selected Febrile Children with Sickle Cell Disease—JA Wilimas, PM Flynn, S Harris, SW Day, R Smith, PJ Chesney, et al. N Engl J Med 1993;329:472.

BACKGROUND: Because of their susceptibility to pneumococcal sepsis, children with sickle cell disease and fever are usually hospitalized for antibiotic therapy. Outpatient treatment may be a safe and less expensive alternative for selected patients. METHODS: After evaluation in the emergency room, children ranging from 6 months to 12 years of age who had sickle hemoglobinopathies and temperatures exceeding 38.5°C were randomly assigned to treatment as either inpatients or outpatients. We excluded from randomization children at higher risk of sepsis (as defined by specific criteria, including temperature above 40°C, white-cell count below 5,000 per cubic millimeter or above 30,000 per cubic millimeter, and the presence of pulmonary infiltrates) or with complications of sickle cell disease (such as a hemoglobin level below 5 g per deciliter, dehydration, or severe pain); these children were treated as inpatients. All patients received an initial intravenous dose of ceftriaxone (50 mg per kilogram of body weight). Those treated as outpatients returned 24 hours later for a second dose of ceftriaxone, whereas the inpatients were treated as directed by their physicians. RESULTS: None of the 86 patients (with a total of 98 febrile episodes) in the randomized groups had sepsis, as compared with 6 of the 70 patients (7 of 86 episodes) excluded because of higher risk ($p = 0.004$). Among the 44 children (50 episodes) assigned to outpatient treatment, there were 11 hospitalizations (22% of episodes) within two weeks after treatment (95% confidence interval, 12 to 36%), whereas after inpatient care only a single patient (2% of episodes) was rehospitalized. When the randomized groups were compared, outpatient treatment saved a mean of $1,195 per febrile episode. The median hospital stay was 3 days (range, 1 to 6) for the children randomly assigned to inpatient care and 4 days (range, 1 to 18) for the higher-risk children treated as inpatients ($p < 0.001$). CONCLUSIONS: With the use of conservative eligibility criteria, at least half the febrile episodes in children with sickle cell disease can be treated safely on an outpatient basis, with substantial reductions in cost.

Plethysmographic Parameters in the Assessment of Reversibility of Airways Obstruction in Patients with Clinical Emphysema—F Gimeno, DS Postma, R van Altena. Chest 1993;104:467.

Slow inspiratory vital capacity (IVC) and forced expiratory volume in 1 s (FEV_1) before and after an inhaled β-agonist are widely used to detect reversible airflow limitation in patients with chronic obstructive lung disease. The measurement of airways

resistance (R_{aw}) during quiet breathing with the body plethysmograph is less frequently used. It may well be of importance in clinical emphysema where measurement of FEV_1 is confounded by the collapse of the bronchi, which does not occur when measuring R_{aw} during quiet breathing. We assessed whether R_{rs}, in addition to IVC and FEV_1, can be used to gain a better insight into the reversibility with 400 μg of fenoterol in patients with clinical emphysema. We studied a group of 51 patients (9 women and 42 men; mean [± SD] age, 64.7 [7.7] years) who had a clinical diagnosis of emphysema. Significant reversibility was identified by spirometry (IVC, FEV_1) and body plethysmography (R_{aw}) in 20 patients (39%). Inspiratory vital capacity alone identified reversibility of airflow limitation in 11 patients (22%). In 5 patients (10%), the postbronchodilator improvement was seen exclusively in the R_{aw} measurement. In the remaining patients, absence of improvement in spirometric and plethysmographic parameters was found. Subjective improvement occurred to the same extent in patients whose R_{aw} and IVC improved. We concluded that R_{aw} gives important information about the reversibility of airways obstruction in patients with clinical emphysema. Therefore, we suggest that tests during quiet breathing should be part of the routine examination of airways obstruction in patients with "irreversible" obstruction by conventional spirometry.

Value of ELISA Using Antigen 60 for the Diagnosis of Tuberculosis in Children—C Delacourt, J Gobin, J-L Gaillard, J de Blic, M Veron, P Scheinmann. Chest 1993;104:393.

We evaluated the possible value of enzyme-linked immunosorbent assay (ELISA) using antigen 60 (A60) for improved diagnosis of tuberculosis in children. Results obtained in 31 children with active tuberculosis and in 16 patients with tuberculous infection without disease were compared with the results of 198 control subjects with no mycobacterial disease. In control children, anti-A60 IgG increased with age and the optical density (OD) in ELISA assays rose from 0.079 ± 0.053 (OD ± SD) in children younger than 5 years old to 0.146 ± 0.082 OD in children older than 5 years. In control subjects younger than 2 years old, IgG OD values were significantly higher in BCG-vaccinated children than in nonvaccinated children. At a chosen specificity of 98%, a positive serodiagnosis was observed in 68% of children with clinically active tuberculosis. In these children with active disease, smears were positive in only 26% of cases and mycobacterial cultures yielded the organism in 45% of cases. None of the infected children without disease had high IgG OD values. IgM measurements were also evaluated. Mean values from control and diseased children overlapped, leading to a low sensitivity (19%) in children with clinically tuberculosis active. We conclude that anti-A60 IgG measurement is a rapid and low-cost technique that enhances the diagnosis of clinically active tuberculosis in children and may distinguish recent infection without disease from infection with disease.

Hemodilution with Oxyhemoglobin: Mechanism of Oxygen Delivery and Its Superaugmentation with a Nitric Oxide Donor (Sodium Nitroprusside)—MW Rooney, LJ Hirsch, M Mathru. Anesthesiology 1993;79:60.

BACKGROUND: Hemodilution (HD) with oxyhemoglobin colloid (oxyHb) provides a greater arterial oxygen content (C_{aO_2}) than HD with conventional colloids; however, oxygen delivery ($\dot{D}_{O_2}$) is essentially the same, because, in contrast to conventional HD, cardiac output (C.O.) is not augmented. This study seeks to elucidate the mechanism that limits C.O. during oxyHb-HD and to test whether infusion of a nitric oxide (NO) donor would augment $\dot{D}_{O_2}$, because oxyHb is known to inactivate *in vitro* endothelial-derived NO. METHODS: Anesthetized dogs were isovolemically hemodiluted with 10% oxyHb, 8% albumin, or 10% methemoglobin (weak NO inactivator) to 20% hematocrit. After HD, sodium nitroprusside (SNP) was titrated intravenously until decreases (> 10 mm Hg) in mean aortic pressure (P_{ao}) indicated the presence of exogenous NO. Systemic hemodynamics and regional blood flows (microsphere method) were measured. RESULTS: Albumin-HD and metHb-HD produced typical HD-mediated responses: increased C.O. (63-65%), slight decreases (13-15%) in $\dot{D}_{O_2}$, decreases in systemic vascular resistance (SVR) proportional to the decreases (49-52%) in blood viscosity of all three groups, and increased regional blood flows (RBF). Responses to oxyHb-HD were atypical: C.O. and its determinants were not changed, $\dot{D}_{O_2}$ decreased (23%) proportional to C_{aO_2}, and SVR and most RBF were not changed except for a net redistribution of C.O. to myocardium and skeletal muscle. In albumin-HD or metHb-HD, SNP (2-5 $\mu g \cdot kg^{-1} \cdot min^{-1}$) induced comparable decreases in mean P_{ao} (29-37%) and SVR (39-41%); however, C.O., RBF, and $\dot{D}_{O_2}$ were not affected. In oxyHb-HD, exceptionally large doses of SNP (54 ± 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$) decreased mean P_{ao} only 19 ± 1%; however, C.O. increased 78 ± 5% and decreases (61 ± 3%) in SVR were slightly greater than viscosity reductions. Other determinants of C.O. were not affected. Most RBF increased proportional to C.O.; there was, however, preferential distribution to myocardium and skeletal muscle. Consequently,

Press and breathe inhalers: For most patients, a juggling act.

The reason:
Pressing and breathing
correctly is not easy.

Studies by specialists show press and breathe inhalers are frequently used incorrectly.

Investigator	No. of patients	No. with incorrect technique
Mayer et al[1]	30	93%
Epstein et al[2]	130	89%
Bailey et al[3]	124	90%
	101	86%
Orehek et al[4]	20	75%

incorrect technique includes the inability of patients to coordinate inspiration with dose delivery.

- Up to 50% of patients revert to their previous, incorrect press and breathe inhaler technique following retraining.[5]
- Even patients who have good technique are inconsistent in applying it, especially during an acute attack.[6]
- **Press and breathe inhalers may provide suboptimal therapy when patients use incorrect technique.**[5,7-10]

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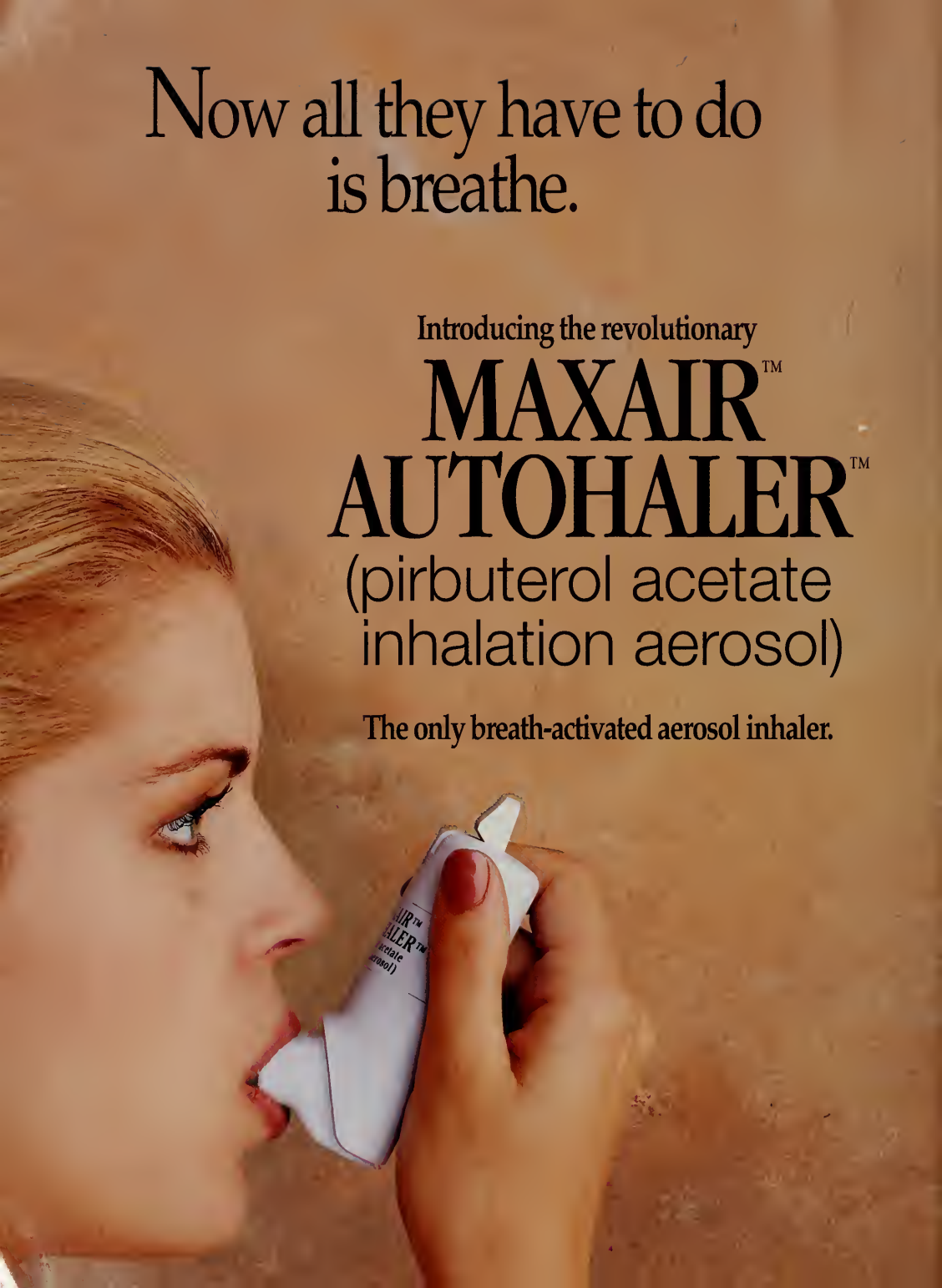


Easier to use correctly and easier to teach than press and breathe inhalers.

Easier to use and teach.

	Press and breathe inhaler (n=70)	AUTOHALER (n=70)
Number able to use inhaler efficiently after reading instructions	27 (39%)	44 (63%)
Number able to use inhaler efficiently after written and verbal instruction	35 (50%)	64 (91%)

In a study of 70 subjects, the number able to use the press and breathe inhaler and Autohaler efficiently after reading the manufacturer's instruction pamphlet, and after reading the instructions reinforced by verbal tuition.

Table adapted from Crompton and Duncan.[11]

Even patients with poor pulmonary function use it correctly.

- In a multicenter study of 156 patients with poor pulmonary function (FEV_1<1.0 L and/or PEFR<200 L), 96.8% were able to activate Autohaler correctly.[12]

Even patients with limited manual dexterity use it correctly.

- In a recent clinical trial, 70% of patients with arthritis (n=48) had little or no difficulty using Autohaler correctly.[13]
- 58% of all arthritis patients (n=48), however, could not operate traditional press and breathe inhalers.[13]

Even elderly patients use it correctly.

- In a study of 21 elderly patients, 71% were able to improve their technique with the breath-activated inhaler.[14]

Maxair™ Autohaler™ provides a consistent dose of medication.

- Maxair Autohaler automatically delivers a metered dose at the right point in the breathing cycle, thus eliminating the need for coordination of pressing and breathing.[15]

Please see last page for summary of prescribing information.

Maxair™ Autohaler™, a more economical way to deliver therapy.

400 inhalations in just one canister.

- ☐ Lower cost per inhalation than Ventolin or Proventil inhalation aerosols.*

With 200 extra inhalations, Maxair Autohaler is like having a built-in refill.†

- ☐ Offers more inhalations than any other leading bronchodilator.
- ☐ Fewer trips to the pharmacy for a prescription refill.

*Prices of Ventolin and Proventil are based on the current published average wholesale prices for one 200 inhalation press and breathe complete unit and one 200 inhalation refill canister. Price of Maxair Autohaler reflects the current published average wholesale price for one 400 dose unit. (Source: Pharmaceutical Data Services, May 1993.) Retail pricing may vary from community to community and may affect cost savings to the patient. Ventolin is a registered trademark of Allen & Hanburys; Proventil, of Schering Corp.

†Please note: Maxair Autohaler is not designed or intended for refill use or for use with other drug canisters.

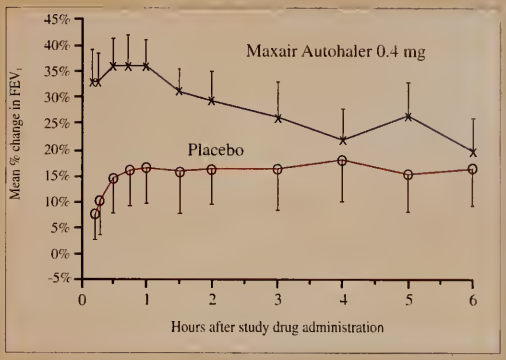
First line for the prevention and reversal of bronchospasm.[16]

Begins working within 5 minutes in most patients.[15]

- The duration of action is maintained for 5 hours in a substantial number of patients. These observations are based on a 15% or greater increase in FEV_1 and were made 5 hours after dosing.[15]

Contains pirbuterol, the newest beta agonist, recognized as similar to albuterol by *The Medical Letter*.[16]

Increased FEV_1.



Mean change in FEV_1 from baseline after administration of placebo aerosol and 400 µg Maxair Autohaler (pirbuterol acetate inhalation aerosol) (n=14).[17]

Low incidence of cardiovascular side effects.[15]

- Studies confirm that pirbuterol produces rapid and sustained bronchodilation with minimal adverse cardiac effects.[18,19]
- Palpitations 1.7%, tachycardia 1.2% (n=761).[15]

As with other beta agonist aerosols, pirbuterol is a sympathomimetic amine and can produce significant cardiovascular effects. Caution should be taken with patients with cardiovascular disorders. The potential for paradoxical bronchospasm and excessive use of inhaled beta agonist drugs should be kept in mind, as these situations can be life threatening. Pirbuterol is not recommended for children under 12 years of age. The adverse events reported for pirbuterol are typical of the beta agonist class of inhaled drugs. The most frequent moderate to severe adverse reactions reported in clinical studies are: nervousness (6.9%), tremor (6.0%), headache (2.0%), dizziness (1.2%), palpitations (1.7%), tachycardia (1.2%), cough (1.2%) and nausea (1.7%).

Please see last page for summary of prescribing information.

NEW BREATH-ACTIVATED

MAXAIR™ AUTOHALER™

(pirbuterol acetate inhalation aerosol)

It helps make the most of aerosol therapy.

The first real advance in aerosol delivery systems in 35 years.

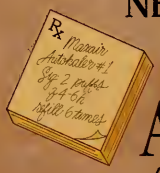
Maxair Autohaler...

- automatically delivers a metered dose at the right point in the breathing cycle, thus eliminating the need for coordination of pressing and breathing.[15]
- is easier to use correctly and easier to teach than press and breathe inhalers.[11]
- provides a convenient alternative to cumbersome spacer devices.[20]
- contains 400 inhalations in every canister, twice that of Ventolin and Proventil inhalation aerosols.

☐ The switch to Maxair Autohaler is easy—it doesn't require a new dosage regimen for your patients who have previously been using a conventional inhaler.

☐ For information relating to medical issues, please call the 3M Pharmaceuticals Medical Services Department at 1-800-328-0255.

Please see next page for summary of prescribing information.



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MAXAIR™
AUTOHALER™
(pirbuterol acetate inhalation aerosol) Bronchodilator Aerosol
For Inhalation Only
BRIEF SUMMARY

INDICATIONS AND USAGE: MAXAIR AUTOHALER is indicated for the prevention and reversal of bronchospasm in patients with reversible bronchospasm including asthma. It may be used with or without concurrent theophylline and/or steroid therapy.

CONTRAINDICATIONS: MAXAIR is contraindicated in patients with a history of hypersensitivity to any of its ingredients.

WARNINGS: As with other beta adrenergic aerosols, MAXAIR should not be used in excess. Controlled clinical studies and other clinical experience have shown that MAXAIR like other inhaled beta adrenergic agonists can produce a significant cardiovascular effect in some patients, as measured by pulse rate, blood pressure, symptoms, and/or ECG changes. As with other beta adrenergic aerosols, the potential for paradoxical bronchospasm (which can be life threatening) should be kept in mind. If it occurs, the preparation should be discontinued immediately and alternative therapy instituted.

Fatalities have been reported in association with excessive use of inhaled sympathomimetic drugs.

The contents of MAXAIR AUTOHALER are under pressure. Do not puncture. Do not use or store near heat or open flame. Exposure to temperature above 120°F may cause bursting. Never throw container into fire or incinerator. Keep out of reach of children.

PRECAUTIONS: General — Since pirbuterol is a sympathomimetic amine, it should be used with caution in patients with cardiovascular disorders, including ischemic heart disease, hypertension, or cardiac arrhythmias, in patients with hyperthyroidism or diabetes mellitus, and in patients who are unusually responsive to sympathomimetic amines or who have convulsive disorders. Significant changes in systolic and diastolic blood pressure could be expected to occur in some patients after use of any beta adrenergic aerosol bronchodilator.

Information for Patients — MAXAIR effects may last up to five hours or longer. It should not be used more often than recommended and the patient should not increase the number of inhalations or frequency of use without first asking the physician. If symptoms of asthma get worse, adverse reactions occur, or the patient does not respond to the usual dose, the patient should be instructed to contact the physician immediately. The patient should be advised to see the illustrated Patient's Instructions for Use.

The Autohaler actuator should not be used with any other inhalation aerosol canister. In addition, canisters for use with MAXAIR AUTOHALER should not be utilized with any other actuator.

Drug Interactions — Other beta adrenergic aerosol bronchodilators should not be used concomitantly with MAXAIR because they may have additive effects. Beta adrenergic agonists should be administered with caution to patients being treated with monoamine oxidase inhibitors or tricyclic antidepressants, since the action of beta adrenergic agonists on the vascular system may be potentiated.

Carcinogenesis; Mutagenesis and Impairment of Fertility — Pirbuterol hydrochloride administered in the diet to rats for 24 months and to mice for 18 months was free of carcinogenic activity at doses corresponding to 200 times the maximum human inhalation dose. In addition, the intragastric intubation of the drug at doses corresponding to 6250 times the maximum recommended human daily inhalation dose resulted in no increase in tumors in a 12-month rat study. Studies with pirbuterol revealed no evidence of mutagenesis. Reproduction studies in rats revealed no evidence of impaired fertility.

Teratogenic Effects — Pregnancy Category C — Reproduction studies have been performed in rats and rabbits by the inhalation route at doses up to 12 times (rat) and 16 times (rabbit) the maximum human inhalation dose and have revealed no significant findings. Animal reproduction studies in rats at *oral doses* up to 300 mg/kg and in rabbits at oral doses up to 100 mg/kg have shown no adverse effect on reproductive behavior, fertility, litter size, peri- and postnatal viability or fetal development. In rabbits at the highest dose level given, 300 mg/kg, abortions and fetal mortality were observed. There are no adequate and well controlled studies in pregnant women and MAXAIR should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers — It is not known whether MAXAIR is excreted in human milk. Therefore, MAXAIR should be used during nursing only if the potential benefit justifies the possible risk to the newborn.

Pediatric Use — MAXAIR AUTOHALER is not recommended for patients under the age of 12 years because of insufficient clinical data to establish safety and effectiveness.

ADVERSE REACTIONS: The following rates of adverse reactions to pirbuterol are based on single and multiple dose clinical trials involving 761 patients, 400 of whom received multiple doses (mean duration of treatment was 2.5 months and maximum was 19 months).

The following were the adverse reactions reported more frequently than 1 in 100 patients **CNS:** nervousness (6.9%), tremor (6.0%), headache (2.0%), dizziness (1.2%). **Cardiovascular:** palpitations (1.7%), tachycardia (1.2%) **Respiratory:** cough (1.2%) **Gastrointestinal:** nausea (1.7%).

The following adverse reactions occurred less frequently than 1 in 100 patients and there may be a causal relationship with pirbuterol **CNS:** depression, anxiety, confusion, insomnia, weakness, hyperkinesia, syncope **Cardiovascular:** hypotension, skipped beats, chest pain **Gastrointestinal:** dry mouth, glossitis, abdominal pain/cramps, anorexia, diarrhea, stomatitis, nausea and vomiting **Ear, Nose and Throat:** smell/taste changes, sore throat **Dermatological:** rash, pruritus. **Other:** numbness in extremities, alopecia, bruising, fatigue, edema, weight gain, flushing.

Other adverse reactions were reported with a frequency of less than 1 in 100 patients but a causal relationship between pirbuterol and the reaction could not be determined: migraine, productive cough, wheezing, and dermatitis.

The following rates of adverse reactions during three-month controlled clinical trials involving 310 patients are noted. The table does not include mild reactions.

PERCENT OF PATIENTS WITH MODERATE TO SEVERE ADVERSE REACTIONS

Reaction	Pirbuterol N = 157	Metaproterenol N = 153	Reaction	Pirbuterol N = 157	Metaproterenol N = 153
Central Nervous System			**Gastrointestinal**		
tremors	1.3%	3.3%	nausea	1.3%	2.0%
nervousness	4.5%	2.6%	diarrhea	1.3%	0.7%
headache	1.3%	2.0%	dry mouth	1.3%	1.3%
weakness	0%	1.3%	vomiting	.0%	0.7%
drowsiness	0%	0.7%	**Dermatological**		
dizziness	0.6%	.0%	skin reaction	0%	0.7%
Cardiovascular			rash	.0%	1.3%
palpitations	1.3%	1.3%	**Other**		
tachycardia	1.3%	2.0%	bruising	0.6%	.0%
Respiratory			smell/taste change	0.6%	0%
chest pain/tightness	1.3%	0%	backache	.0%	0.7%
cough	.0%	0.7%	fatigue	.0%	0.7%
			hoarseness	.0%	0.7%
			nasal congestion	0%	0.7%

OVERDOSAGE: The expected symptoms with overdosage are those of excessive beta-stimulation and/or any of the symptoms listed under adverse reactions, e.g., angina, hypertension or hypotension, arrhythmias, nervousness, headache, tremor, dry mouth, palpitation, nausea, dizziness, fatigue, malaise, and insomnia.

Treatment consists of discontinuation of pirbuterol together with appropriate symptomatic therapy.

The oral acute lethal dose in male and female rats and mice was greater than 2000 mg base/kg. The aerosol acute lethal dose was not determined.

CAUTION: Federal law prohibits dispensing without prescription.
Store between 15° and 30°C (59° to 86°F).
For Full Prescribing Information, see package insert.

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the augmented C.O., and C_{aO_2} of oxyHb-HD, produced large increases in $\dot{D}_{O_2}$, 77 ± 5% from HD alone and 43 ± 3% from prehemodilution values. CONCLUSIONS: This study indicates that the limited C.O. and $\dot{D}_{O_2}$ of oxyHb-HD resulted from opposing changes in two determinants of flow, ie, reduced blood viscosity and increased arterial resistance (vasoconstriction). The vasoconstriction was not evident with metHb-HD and was reversed by the SNP infusion, indicating that oxyHb inactivated in vivo endothelial-derived NO. The ability of the NO donor (SNP) to facilitate large viscosity-mediated increases in $\dot{D}_{O_2}$ during oxyHb-HD is an important finding that could potentially render oxyHb colloids more useful than conventional colloids, particularly for the individual with a compromised circulation who would benefit from an increased oxygen supply.

Smoking and Pregnancy Outcome: Trends among Black Teenage Mothers in Missouri—GH Land, JW Stockbauer. Am J Public Health 1993;83:1121.

OBJECTIVES: The purpose of this study is to analyze the smoking changes that have occurred among pregnant Black teenagers in Missouri. The study also examines changes in Black teenage pregnancy outcomes in relation to smoking behavior changes. METHODS: This analysis used computerized data files from the 1978 to 1990 Missouri birth certificates to acquire information on smoking during pregnancy for 41,544 Black teenagers and 105,170 White teenagers. All Missouri births with smoking history were included in the study. RESULTS: During the study period, the rate for Blacks who smoked during pregnancy decreased from 37% in 1978 to less than 22% in 1990. A large part of this reduction is attributable to Black teenagers, whose smoking-during-pregnancy rate declined from 35.8% to 7.2%. Additionally, the Black teenage-specific low-birthweight rate decreased by 13.6% over the study period, possibly influenced by the decrease in smoking. CONCLUSIONS: The results indicate that a major norm has changed in smoking status among pregnant Black teenagers. Understanding the reasons behind this change could assist smoking cessation and other health promotion efforts.

The Optimal Time for Measuring the Cardiorespiratory Effects of Positive End-Expiratory Pressure—M Patel, M Singer. Chest 1993; 104:139.

No study has elucidated the minimum time taken for oxygen delivery to reach steady-state levels after an increase in positive end-expiratory pressure (PEEP). We therefore investigated 10 ventilated patients who received a total of 27 increments in PEEP of 5 cm H_2O. Cardiorespiratory parameters were measured at baseline, at 1 min, and thereafter at 5-min intervals until 2 consecutive measurements of oxygen delivery were constant. Cardiac output was measured continuously using an esophageal Doppler transducer. The PEEP was increased to a maximum of 20 cm H_2O or until oxygen delivery fell by 15%. Increases in PEEP were always associated with increases in oxygen saturation. Cardiac output decreased in the majority of cases, the greatest falls occurring in the first minute after an increase in PEEP, and with higher levels of PEEP. No significant changes in oxygen delivery occurred after 15 min. We therefore recommend that 15 min be allowed to elapse after an increase in PEEP before oxygen delivery is reassessed.

Does Implementing Pulse Oximetry in a Critical Care Unit Result in Substantial Arterial Blood Gas Savings?—KJ Inman, WJ Sibbald, FS Rutledge, M Speechley, CM Martin, BJ Clark. Chest 1993;104:542.

OBJECTIVES: To examine the impact of pulse oximetry on the use of arterial blood gas and other laboratory determinations and to examine predictors of the use of arterial blood gas measurements. DESIGN: Before (preoximetry)/after (postoximetry) study. SETTING: Thirty-bed multidisciplinary critical care unit. PATIENTS: Consecutive admissions of 300 patients (150 before and 150 after oximetry). MEASUREMENTS: For each patient examined, the number of arterial blood gas determinations, serum electrolyte levels, complete blood chemistries, arterial lactate levels, and creatinine samples were recorded for the initial 9 days of the stay in the critical care unit. These data were stratified by nursing shift (day vs night) and by the source of the admission (medical vs surgical). Other information collected included demographic variables, the severity of illness, the length of stay in the critical care unit, and various ventilatory parameters. RESULTS: Introducing pulse oximetry was associated with a marginal (10.3%; $p < 0.025$) reduction in the use of arterial blood gas determinations. This decrease was accounted for by changes occurring on the night shift and in the surgical patient. These findings were also observed for serum electrolyte determinations. No significant differences in the use of arterial blood gas measurements were found for medical patients. No significant differences were found in the use of arterial lactate levels, complete blood chemistries, or creatinine determinations. Significant predictors of arterial blood gas determinations included the number of days intubated, the number of ventilator orders, the num-

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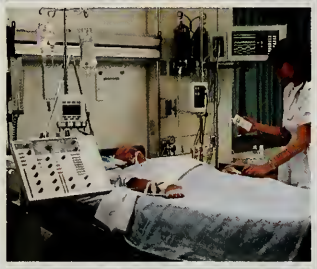

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ber of days on an inspired oxygen content (F_{IO_2}) greater than 50%, and the acute physiology and chronic health evaluation II (APACHE II) score. CONCLUSIONS: The implementation of pulse oximetry in this manner gives an idea how effective the technology will be in reducing the use of arterial blood gas determinations without guidelines for the use of pulse oximetry. As only a marginal decrease was observed in the total population of medical and surgical patients, and only on the night shift, formal and standardized guidelines for the most efficient use of pulse oximetry should be considered. If these were considered, pulse oximetry may indeed make a significant contribution to improving the efficiency of care services.

Comparison of BiPAP Nasal Ventilation and Ventilation via Iron Lung in Severe Stable COPD—T-C Lien, J-H Wang, M-T Chang, C-D Kuo. Chest 1993;104:460.

The effects of noninvasive ventilators on COPD remain controversial because of their obscure mechanisms. A randomized crossover study, using iron lung and positive pressure nasal ventilation (BiPAP) each for 40 min, was per-formed in 11 stable patients with severe COPD. Throughout the study, we monitored surface EMGdi, EMGst, ECG, S_{aO_2}, ET_{CO_2}, and the movements of RC and AB. Afterwards the data were replayed to calculate V_T, RR, pulse rate, V_T/T_I, iEMG, and phase angle. No statistically significant improvement was found in view of the above parameters. However, the percentage of iEMGst change after 40-min BiPAP ventilation, compared with the baseline, was much more significant in patients with FEV_1 below 0.55 L than those with FEV_1 above 0.55 L (n = 4:7, iEMGst –62.93% ± 23.27% vs 32.45% ± 42.79%, p = 0.0056). iEMGst correlated significantly with FEV_1 during BiPAP ventilation ($p < 0.05$, $r = 0.59$). We conclude that the iEMGst during short-term BiPAP ventilation correlates with the severity of the disease.

Health Effects and Exposure Assessment of Aerosolized Pentamidine Handlers—MA McDiarmid, J Fujikawa, J Schaefer, G Weinmann, RE Chaisson, CA Hudson. Chest 1993;104:382.

Nurses administering aerosolized pentamidine (AP) were studied to determine any effect AP may be having on their health. Exposure was determined by each nurse's self-report of treatment given as recorded in a daily log and personal and area pentamidine sampling. Outcome measures were self-reported symptoms recorded in a daily log and peak expiratory flow rates (PEFR) and cross-shift and cross-week pulmonary function tests (PFTs). Results revealed no dose-response effect of pentamidine exposure on cross-shift and cross-week PFTs. However, declines in cross-shift PEFRs, diffusion capacities, and increased symptom complaints were observed for a subset of the study population. This suggested that outcomes were modulated by host factors (history of hay fever and allergy) as well as exposure doses. Treatment booth efficacy in containing fugitive AP aerosol was also corroborated as a means of minimizing worker exposure.

Positive End-Expiratory Pressure Lowers the Risk of Laser-Induced Polyvinylchloride Tracheal-Tube Fires—AG Pashayan, C SanGiovanni, LE Davis. Anesthesiology 1993;79:83.

BACKGROUND: The possibility of ignition of polyvinylchloride (PVC) tracheal tubes by a CO_2 laser is of concern in patients undergoing CO_2 laser surgery of the airway. The authors analyzed the ignition of PVC tracheal tubes by a CO_2 laser beam to determine what variables were involved, and then designed a study to determine how they affect the incidence of such fires. METHODS: For the analysis, PVC tracheal tubes were enclosed in a clear plexiglass enclosure and a laser beam was focused on the tubes. The enclosure contained one of three different gas combinations. A high-speed camera photographed the tubes during the analysis and showed that tracheal tube perforation always preceded ignition in all three gas combinations. These results led to the hypothesis that intraluminal gauge pressure (IGP) may be an important variable, because it would affect the flow of O_2 across the perforation. This hypothesis was tested by aiming a CO_2 laser beam at PVC tracheal tubes and varying IGP in 0.25-cm H_2O increments, from 0.25 to 28 cm H_2O, while nitrogen (N_2) or helium (H_e) containing O_2 at 40, 50, or 60% flowed through the tubes. To simulate the clinical effect of IGP on PVC tracheal tube ignition, we used a mechanical lung model connected to an anesthesia breathing circuit with a standing bellows ventilator in which 60% He and 40% O_2 flowed through a PVC tracheal tube. Laser beam exposure was started at three different times during the respiratory cycle: at the start of inspiration, at the end of inspiration, or at the end of expiration. Also, for each condition, trials were made at haseline circuit pressure (2.5 cm H_2O) and at 5.0 cm H_2O by the addition of 2.5 cm H_2O positive end-expiratory pressure (PEEP) applied to the circuit. RESULTS: The incidence of tracheal tube ignition decreased as IGP increased. The IGP at which ignition did not occur (which increased as O_2 concentration increased) did not dif-

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fer between N_2 and He at 40% O_2, but was twice as high with N_2 as with He at O_2 of 50% and 60%. Fires never occurred when PEEP was added to the system and, when PEEP was not added, always started during the last 2 s of end expiration (when airway pressure is lowest), regardless of when the laser beam was activated. CONCLUSIONS: It is recommended that, in addition to other safety practices, PEEP be added to the breathing circuit during CO_2 laser operations on the airway in which PVC tracheal tubes or laser-resistant tracheal tubes with PVC components are used.

The Lack of Effect of Routine Magnesium Administration on Respiratory Function in Mechanically Ventilated Patients—D Johnson, C Gallagher, M Cavanaugh, R Yip, I Mayers. Chest 1993;104:536.

STUDY OBJECTIVE: We wished to determine if magnesium infusion would improve respiratory muscle function in long-term ventilated patients even in the absence of hypomagnesemia. DESIGN: Prospective study of mechanically ventilated patients using a double-blind crossover design. SETTING: A combined medical-surgical ICU of a university teaching hospital. PATIENTS: 21 separate admissions to the ICU in 20 patients were studied. Patients who were selected had been intubated and mechanically ventilated for at least 6 days with the admitting diagnosis of respiratory failure. INTERVENTIONS: Twelve patients received 6g $MgSO_4$ intravenous (I.V.) infusion over 16 h on Day 1 followed by placebo infusion on Day 2. Nine patients received placebo on Day 1 followed by $MgSO_4$ (6 g I.V.) on Day 2. MEASUREMENTS & MAIN RESULTS: We measured vital capacity (VC), maximal inspiratory pressure (P_{Imax}), and maximal expiratory pressure (P_{Emax}) in all patients. There were no significant differences in P_{Imax} (37 ± 14 vs 42 ± 20 cm H_2O), P_{Emax} (59 ± 32 vs 61 ± 38 cm H_2O), and VC (850 ± 460 vs 960 ± 490 mL) comparing values before and after magnesium infusion. We could not find a subgroup of patients with a marked improvement in P_{Imax} or P_{Emax}. CONCLUSIONS: In patients requiring mechanical ventilation for respiratory failure, magnesium infusion is not associated with increased respiratory muscle strength. Although a trial of $MgSO_4$ administration may be considered for patients with difficulty weaning from mechanical ventilation, it is unlikely to result in clinical improvement.

Contribution of Water Condensation in Endotracheal Tubes to Contamination of the Lungs—P Cardinal, P Jessamine, C Carter-Snell, S Morrison, G Jones. Chest 1993;104:127.

We postulated that water condensate in endotracheal tubes (ETTs) transports bacteria in the ETTs into the lungs during mechanical ventilation. Thirty-two ETTs obtained from freshly extubated patients were studied under wet and dry conditions using a physiologic lung model. All bacteria expelled from the ETTs were collected on culture plates positioned beneath the ETT. The lung model was ventilated with saturated air at 37°C over two time periods (60 min each), one in which condensation formation was prevented and the second in which condensation formed within the ETT. A mean of 157.6 colony-forming units (CFU)/h were expelled with condensation compared to a mean of 2.4 CFU/h without condensation. We concluded that bacteria were continuously transported from the ETT into the lungs during mechanical ventilation in water droplets. Prevention of water condensation abolishes this constant bacterial inoculation in a lung model.

Preoperative Prediction of Pulmonary Complications following Thoracic Surgery—RE Dales, G Dionne, JA Leech, M Lunau, I Schweitzer. Chest 1993;104:155.

The ability of preoperative quality-of-life and physiologic variables to predict postoperative complications was tested in 117 consecutive patients undergoing thoracotomy for possible or definite lung cancer. Preoperatively, quality of life was globally assessed by the QLI and Sickness Impact Profile. Dyspnea was assessed by the Clinical Dyspnea Index and a modified Pneumoconiosis Research Unit question. Spirometry and maximal exercise testing were carried out in 115 and 46 subjects, respectively. Thirty-seven percent experienced at least one respiratory complication (eg, pneumonia, atelectasis prompting bronchoscopy, pulmonary embolism). Twofold or greater increases in respiratory complications were associated with current smoking ($p < 0.05$), cancer as the final pathologic condition ($p < 0.10$), at least moderate dyspnea ($p < 0.10$), $FEV_1 < 60\%$ of predicted ($p < 0.05$), ventilatory reserve < 25 L ($p < 0.05$), and $V_{O_2max} < 1.25$ L ($p < 0.05$). Twofold increases in the incidence of any complication (respiratory, cardiac, etc) were associated with age ≥ 75 years ($p < 0.05$) and cancer as the final pathologic condition ($p < 0.05$). We conclude that simple historic information (age, smoking status, cancer status, dyspnea) indicates the risk of postoperative morbidity. General quality-of-life measures were not good predictors of morbidity. Our findings corroborate the few studies supporting the value of V_{O_2max} and suggest that the usefulness of the ventilatory reserve deserves further attention.

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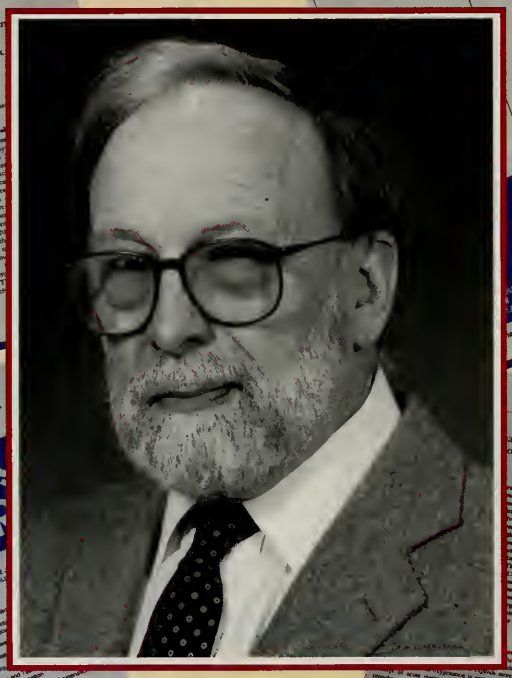

Many people have worked diligently to advance the respiratory care profession, but even among those many Philip Kittredge's vision, constancy, and unwavering commitment are unparalleled. Without that vision, constancy, and commitment, there would be no Journal and there might be little scientific foundation for respiratory care. Phil retired from the Journal's editorial staff on June 30, 1993. For 31 years of service to the profession—25 of those as editor of RESPIRATORY CARE—**we thank you, Phil!**

Walking the Walk—Not Just Talking the Talk: Cigarette Smoking and Respiratory Care Practitioners

Cigarette smoking is recognized as the single most important cause of preventable morbidity and mortality in our country, and health promotion and disease prevention efforts of recent years have brought pressure to bear on those who continue to smoke.

Like it or not, healthcare professionals—particularly respiratory care practitioners (RCPs)—are role models for patients and families seeking to modify or defend their smoking behaviors.

We have observed that some RCPs continue to smoke despite firsthand knowledge of the health hazards, despite their conspicuous position in caring for patients with lung disease, and despite their professional obligation to be positive role models and nonsmoking advocates. To us, smoking among healthcare professionals and in particular among RCPs—the group who cares each day for patients suffering from the complications of cigarette smoking—is the antithesis of professional behavior.

RCPs with their in-depth knowledge of respiratory physiology and pathology and firsthand experience with the devastating effects of smoking have a responsibility to be role models—positive role models—for those seeking to stop smoking.

We reviewed the literature of the last 5 years to ascertain the prevalence of smoking behavior and attitudes toward this behavior among health professionals. We found 6 studies of nurses,[1-6] 1 of dental hygienists,[7] 1 of Ethiopian healthcare professionals,[8] and 1 of Australian physicians.[9] We found no studies quantifying the amount and nature of smoking or attitudes toward smoking among RCPs.

The six studies of the smoking habits of nurses conducted since 1986 have documented smoking behavior of 20-27.2%, with a mean of 23.3%.[1-6] Earlier researchers (1973-1983) reported that 25-29% of nurses smoked cigarettes.[10-15] Smoking prevalence among nurses in trending downward, and the mean is now lower than the 1992 Centers for Disease Control and Prevention report of 25.5% for the general public (1990 data).[16] Physician smoking rates have decreased in a similar manner but at a more rapid rate, from 36% in 1979,[17] 10% in 1983,[18] 16.7% in 1986,[19] and 8% in a study of oral surgeons in 1987.[20]

Because no studies of smoking behavior or attitudes toward smoking among respiratory therapists were found, The American Lung Association of Delaware and Chester Counties in Pennsylvania (ALA) undertook a survey, limited to one district of the Pennsylvania Society for Respiratory Care. Of 1,582 surveys sent to directors of hospital respiratory care departments to be distributed to RCPs, 511 were returned (32.4%). The geographic limitation, nonrandom sampling technique, and the low response rate allow no generalization to other groups or to the population of U.S. respiratory therapists; however, the results (as yet unpublished) are the only data available at the present time on smoking prevalence among RCPs.

Of the 511 ALA respondents, 25% are currently smoking, 29% are former smokers, and 46% have never smoked. The smoking rate of 25% among this group falls within the range of the smoking nurses, is higher than the mean, and is only 0.5% lower than that of the general public. This finding is contrary to the supposition advanced by Haughey et al (1989)[6] that a low smoking rate in oncology nurses might be related to their exposure to the health consequences of smoking. If this explanation is correct, RCPs would have a lower rate of smoking compared to groups with less awareness of the consequences. In our sample, this is not the case. Further, RCPs and nurses have not given up smoking in numbers as great as have physicians.

The differences in smoking prevalence between nurses, therapists, and physicians may be related, at least in part, to education level. The ALA survey found significant differences among smokers, nonsmokers, and former smokers based on the credential they had earned ($p < 0.001$) and the degree they had attained ($p < 0.003$). This finding is similar to the common finding of Becker et al (1986),[1] Haughey et al (1989),[6] and Faehnrich & Gerlach (1989)[5] that nurses with less education had a higher rate of smoking. The 1988 Centers for Disease Control and Prevention report found that smoking in the general population was greatest in the least-educated Americans.[21]

Of the smokers in the ALA survey, 76.8% have tried to quit and 57.5% are interested in quitting. Although it is encouraging that approximately one half of the RCP smokers want to quit, it is dismaying to learn that so many have tried and failed. Of the former smokers who successfully quit, 63% used the 'cold turkey' method. Therefore, the desire to quit must be a key factor in successful quitting. Once a smoker has the desire, the question becomes, What are the variables that make some successful while others continue to smoke? Researchers have found differences in smoking behavior among nurses, based on workplace,[2,4] age, sex,[1] and rotating shifts,[4] but the ALA survey did not find significant differences among the RCP groups based on any of these factors.

Other cited reasons for continuing to smoke include social and psychological pressures and nicotine dependence. In the ALA survey, a discriminant analysis using nicotine dependence, psychological and social factors, and credential and degree to predict membership in the smoker or former smoker categories found that only nicotine dependence, credential, and degree were significant ($p < 0.002$). A regression analysis using the same factors as the discriminant analysis to predict smoking behavior (ie, number of cigarettes smoked) was significant for nicotine dependence ($p < 0.001$). These findings agree with those of DeMello et al (1989)[3] in which the only significant differences between smokers and former smokers were in the responses to statements concerning nicotine dependence. No conclusions can be drawn as to whether the instrument used measured an actual physical dependence or a perceived physical dependence. However, dependence on nicotine (perceived or physiologic) plays an important role in the continued smoking behavior of the RCPs surveyed.

Several researchers have explored the attitudes of nurses[3,4,6] and dental hygienists[7] toward their position as role model for nonsmoking behavior. The majority of respondents in each study agreed that they should set a good example by not smoking. However, Fried & Rubinstein (1990)[7] and Dore & Hoey (1988)[4] found that nonsmokers were more likely to agree with the role-modeling statement than were smokers. The ALA findings were consistent with this, showing 74.8% of the smokers and 85.7% of the former smokers agreeing with the statement. A statistically significant difference existed between smokers and former smokers in response to the statement, "Respiratory therapists should set a good example by not smoking cigarettes" ($p < 0.001$). RCPs who smoke may not believe that positive role modeling of nonsmoking behavior is an important component of their professional responsibility. This is analogous to a nutritionist who believes that overeating and unhealthy eating are acceptable habits. It is difficult to believe that an overweight dietitian has professional credibility. In a similar manner, it is difficult to believe that an RCP who smokes can maintain professional credibility.

Previous studies[1,3-7] have also explored attitudes toward the role of encouraging patients to stop smoking. The majority of respondents agreed that nonsmoking advocacy should be a part of their job responsibilities. A comparison of smokers and nonsmokers showed that more nonsmokers or former smokers agreed than smokers.[1,4] This is consistent with the ALA survey findings; more former smokers (88.4%) agreed with nonsmoking advocacy than smokers (81%). A statistically significant difference existed between former smokers and nonsmokers in response to the statement, "Respiratory therapists should actively encourage patients to stop smoking" ($p < 0.001$). Smoking behavior may interfere with a therapist's ability to effectively perform in health-promotion activities. How credible is a professional who continues to smoke while attempting to help a patient to quit smoking?

Although the ALA study has limitations, it raises interesting questions and concerns regarding the role-modeling behavior of RCPs who smoke. If smoking prevalence actually approaches that of the

general public, as has been suggested by the ALA study, the image of our profession as a whole is at risk. RCPs should be at the forefront in smoking cessation—their own! We believe that the time has come for the profession to take a stand and direct attention toward eliminating smoking within its own ranks!

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Supported by the ALA of Delaware and Chester Counties and The Bryn Mawr Hospital.

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Adventures in Recycling: The Reuse of "Disposable" Pulse Oximeter Probes

A billion seconds ago, Don Larsen was pitching a perfect game in the World Series. A billion minutes ago, Hannibal was crossing the Alps with his troops. A billion hours ago, the Earth was a cold, solid piece of rock. And a billion dollars is what the United States spent on health care since 9 AM yesterday.

Senator Alan K Simpson, R-Wyoming [1]

Much has been said and written about the need for measures to control costs in the American healthcare system. Unfortunately, the only thing that is cheap any more in healthcare is talk. But, the time has come for doing much more than talking. Assumptions must be challenged, and creative ways of managing resources must be developed.

On numerous occasions, we have spoken with physicians from other countries who often comment on the vast amount of waste in the U.S. healthcare system. With this in mind, it is time for us in the respiratory care community to critically review all of our patterns of resource consumption.

One of the most conspicuous (and, in our opinion, indefensible) of these patterns is the routine use of disposable pulse oximeter probes. In our facility, we consume about 12,500 disposable pulse oximeter probes per year for a total cost to the hospital of approximately $168,000.00. Charges to patients for these supplies are approximately $237,500.00 yearly. When we began to look for ways to effect some serious savings, we considered how to reduce probe costs. Of course, one way would be to reduce the overall use of pulse oximetry—something that we have done with some success.

We then asked (1) Could we use a nondisposable probe or (2) could "disposable" probes be reused?

The use of nondisposable probes is not a suitable alternative in our facility because the design of the brand of permanent probe that we would use is not optimal for or adaptable to broad neonatal and pediatric applications.

When we began to consider reusing disposable probes, we were, at first, not very optimistic because we had heard from a number of people (who ought to be well informed about such things) that the reuse of items marked "single-patient use" was not possible. We were told that reuse would be illegal (ie, against government rules or regulations) and dangerous (ie, contribute to the incidence of nosocomial infections) and that—in the case of pulse oximeter probes—they probably wouldn't work if reused, and besides the hospital wouldn't allow reuse anyway because it was against hospital policy.

However, one of our principal operating philosophies is to challenge assumptions, and thus we set out to discover if the impediments to reusing "single-patient use" items could be removed—if in fact those impediments existed at all.

A review of the current government regulations and recommendations regarding reuse revealed the following statement from the Centers for Disease Control and Prevention, "the recommendation against reprocessing and reusing single-use items has been removed."[2] In fact, a review of the literature revealed a number of descriptions of the safe reuse of disposable items.[3-13] The interested reader is directed to the excellent reviews of these issues by Chatburn[5] and Greene.[14] In short, there appear to be no regulatory impediments to reuse of supplies labeled 'single patient use.'

Indeed, a prominent pulse oximeter probe manufacturer now offers a disposable pulse oximeter recycling program that is designed to allow users to return used disposable oximeter sensors to the manufacturer and then to buy back the same probes after reprocessing—at a slightly reduced price.[9]

Reuse of items marked "single patient use" has been occurring in many hospitals for many years. Some of the items reused include disposable syringes, cardiac catheters, hemodialyzers, ventilator circuits, endotracheal tubes, tracheal tubes, orthopedic appliances, suture-staple removers, cautery devices, esophageal thermometers, ear syringes, face tents, gastric pH monitors, hypodermic needles, Javid tubes, oxygen masks, microscalpels, stone baskets, surgical gloves, surgical packs, tri-adaptors, urethral stints, and urinary bags.[14] Reports indicate that from 41 to 58% of hospitals in North America are reusing disposable devices[14,15]—and those reused devices include disposable pulse oximeter probes.[9]

What about the second caveat—that reuse of disposable pulse oximeter probes might be dangerous inasmuch as it could contribute to infection transmission between patients? According to the Centers for Disease Control and Prevention "There is a lack of evidence indicating increased risk of nosocomial infections associated with the reuse of all single-use items."[2] Certainly, this would seem to be true of items that are noninvasive and touch only intact skin (such as pulse oximeter probes). These rarely if ever cause disease.[2] With these facts in mind, we felt the reuse of disposable pulse oximeter probes was probably safe from an infection-control point-of-view; however, as an added precaution, we decided that a recycling program would include gas sterilization of all probes to be reused.

The third caveat against the reuse of these probes was the concern about the saturation data they would produce. In order to peform a preliminary test of the effects of reprocessing, we gathered 50 discarded disposable pulse oximeter probes that had been used on patients. Figure 1 illustrates how the adhesive material affixed to the probe by the manufacturer was cut off. Following this, the probes were gas autoclaved and new tape was applied.

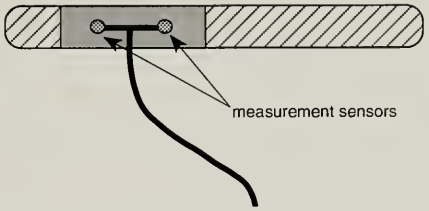

Fig. 1. Preparation of a disposable pulse oximeter probe for reprocessing. Striped area is cut off after each patient use.

To test these probes, we compared their performance against that of a new probe. A new pulse oximeter probe was applied to the small finger of one hand and then attached to a widely used pulse oximeter. Data for heart rate and saturation were recorded and good signal quality ascertained by viewing the plethysmographic display in the typical clinical fashion. Then, a reprocessed probe was immediately applied to the small finger of the other hand. Using the same oximeter, the new probe was disconnected, the reused probe connected, and the data acquired in the same fashion. A sample of 50 reprocessed probes was tested. Mean heart rate was found to be 78 ± 1.2 beats per minute when using a new probe and 79 ± 1.6 beats per minute when using reprocessed probes ($p = 0.83$, Student's t test). Mean oxyhemoglobin saturation by pulse oximeter (S_{PO_2}) was found to be 95 ± 1.2 % when using a new probe and 94.7 ± 1.6 % when using reprocessed probes ($p = 0.83$). Thus, we were convinced that—at least after a single episode of reprocessing and with a normal subject—reused disposable pulse oximeter probes produced data not significantly different from a new probe.

Finally, a review of our hospital's internal regulatory literature contained a provision for the reuse of single-patient-use items so long as the program had the approval of the infection control committee. So we set out to implement a program to reuse disposable pulse oximeter probes.

Some keys to a safe and effective reuse program are that it (1) be defined in a written policy, (2) contain specific methodology for testing the function of devices after reprocessing, and (3) contain specific methods for obviating the risk of cross-infection of patients.

We are satisfied (at this point) that our program meets these requirements. We have written a position paper describing our rationale for reuse and a departmental policy and procedure detailing how probes would be gathered, inspected, sterilized, tested, and packaged. These details were submitted to the hospital risk manager, patient care services administrator, chief of the medical staff, infection control committee, and department medical director, all of whom approved the program. We then developed a hospital-wide education program for nursing and medical staff that included a poster (Fig. 2) describing the recycling program. This poster was duplicated and displayed at every nursing station. Additionally, we attached memoranda describing the program to each nursing service paycheck. The importance of a broadly administered, ongoing education program for nurses and physicians cannot be overemphasized.

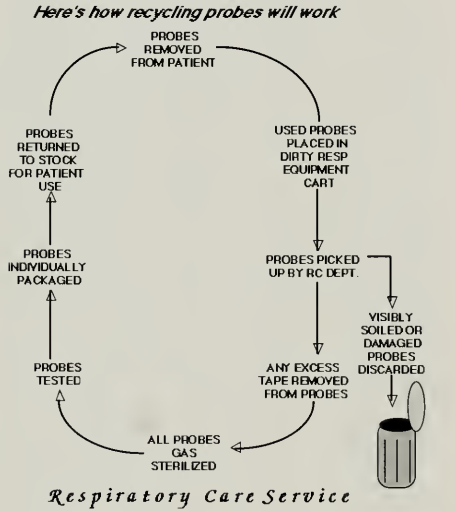
Here's how recycling probes will work
PROBES REMOVED FROM PATIENT
USED PROBES PLACED IN DIRTY RESP EQUIPMENT CART
PROBES PICKED UP BY RC DEPT.
VISIBLY SOILED OR DAMAGED PROBES DISCARDED
ANY EXCESS TAPE REMOVED FROM PROBES
ALL PROBES GAS STERILIZED
PROBES TESTED
PROBES INDIVIDUALLY PACKAGED
PROBES RETURNED TO STOCK FOR PATIENT USE
Respiratory Care Service

Fig 2. Poster used to inform the nursing staff about the pulse-oximeter probe-recycling program.

Because many clinicians habitually throw materials away, we were concerned that probes would not be returned for reprocessing. We expained to our respiratory care practitioners (RCPs) that the financial performance of the department (and hence long-term job security of all) was influenced by this program. Respiratory care practitioners have done an excellent job of capturing and returning used probes; nurses tend to leave the probe attached to the monitor when it is discontinued, returning the probe and the monitor to the dirty equipment room. We have encouraged this practice because it improves probe capture—and because it is convenient, they tend to do it most of the time.

All probes are visually inspected after use, either by a nurse, an RCP, or an equipment technician. Visibly soiled probes and obviously damaged probes are discarded. The probe manufacturer has expressed concerns about the quality of the data produced by recycled probes. To this we respond: (1) we continuously test and record the quality of the data obtained from recycled probes, and to date have noted no clinically or statistically important differences, (2) some number of brand new probes don't work when taken 'out of the box.' Our impression is that the bad-probe rate is similar between new and recycled probes. We have, to date, processed and reused over 1,400 probes, many of them more than once, and have yet to encounter a serious clinical problem. Probes are reprocessed repeatedly until they (1) are visibly soiled, (2) are visibly damaged, (3) or cease to function.

Further, we believe that any clinician who is unable to determine whether data from a pulse oximeter are of acceptable quality probably shouldn't be using oximetry in the first place.

Because our test of reprocessed probes only includes saturations in a normoxic range, it might be argued that we are unable to assure the accuracy of reprocessed probes in profoundly desaturated patients. Although we did not test this question, it has been our clinical experience that brand new probes do not confer on the user any additional sense of certainty that low readings are accurate. A paucity of scientific evidence exists regarding the performance of various types of new disposable probes on hypoxic patients. Finally, we continue to be confused about the clinical importance of whether an oximeter reading is 'really' 65% or 75% because in either case the patient will be treated aggressively for hypoxia.

It is an interesting dichotomy that many pulse oximeter manufacturers have long warned us not to rely entirely on the readings of their instruments to manage patient's oxygenation, and yet have si-

multaneously saturated (no pun intended) the intensive-care market to the point where a pulse oximeter is found on almost every mechanically ventilated intensive-care patient.

Of our stock available for use at any given moment, approximately one third have been processed more than once, and one fourth more than twice. To date, we have discovered 12 reprocessed probes that failed to work when attached to the oximeter (the monitor indicated a probe malfunction), yielding a failure-rate of 0.86%. While we have never systematically collected data on the failure-rate of new probes, it is the consensus of our staff that the failure rate of recycled probes is equal to or less than that of new probes.

Questions that remain to be answered include How many times can a disposable pulse oximeter probe be reused safely? At this time we do not intend to arbitrarily limit the number of times a probe can be reused, but instead to follow our previously described guidelines.

One frequent criticism of recycling programs is that they are labor-intensive and thus not necessarily cost-effective. Because we spend more than $160,000 each year on pulse oximeter probes, it is hard to imagine how we could not save money on a recycling program. During the initial phase of this program, we incurred no labor costs due to recycling because clinical managers, hospital volunteer staff, and office staff all pitched in to see how well such a program would work. From experience, we estimate that 8-12 hours/week are required to recycle pulse oximeter probes. We have created such a position. At $7.00/hour, this equates to $84/week or $4,368/year—the cost of 312 pulse oximeter probes. We estimate that in the first 12 months of this program, we will spend $50,000 for pulse oximeter probes, approximately $117,000 less than we would spend without recycling (assuming no price increase in probes). The cost of gas autoclaving is minimal because we typically gas a batch of several hundred probes at a time at a cost of about $70/batch.

We estimate the cost of recycling a single probe to be $0.23 for labor, and $0.40 for materials and autoclaving. Compare this $0.63/probe to the manufacturer's charge of $14.00 for a new probe.

We have now been running our recycling program for about 3 months and have not yet received a serious complaint. Interviews with staff members yield consistently positive responses to the program.

We encourage all respiratory care services to critically evaluate their resource consumption patterns and consider recycling as a less costly alternative.

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New Rules, New Roles, New Responsibilities

American Association for Respiratory Care 39th Annual Convention and Exhibition
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MEDWATCH: The New FDA Medical Products Reporting Program

The Food and Drug Administration (FDA) with the support of over 70 health professional and industry organizations recently launched a new medical products reporting program called MEDWATCH.[1] MEDWATCH is designed for use by health professionals to let the FDA quickly know about serious adverse events and product problems that occur with drugs, biologics, medical devices, and special nutritional products.

Reports from individual physicians and other health professionals are crucial to a successful national postmarketing surveillance program. For example, reports submitted to the Agency, either directly or via the manufacturer, played a critical role in the identification of a serious cardiac arrhythmia (torsade de pointes) associated with overdose of terfenadine and astemizole and their interactions with ketoconazole and the macrolide antibiotics.[2] Other reports led to the discovery of paradoxic bronchospasm[3] associated with the use of inhaled beta-agonist agents and the occurrence of gas gangrene with the use of Susphrine injection (unpublished data, Div of Oncology and Pulmonary Drug Products, Center for Drug Evaluation and Research, FDA, Rockville MD).

An important product problem was uncovered through reporting when a manufacturing change in which soya was added to the formulation of metaproterenol caused choking in patients. Subsequent reformulation by the manufacturer removed the soya preservative (unpublished data, ibid).

Reports from health professionals also prompted the FDA to recently issue a Safety Alert warning of instances in which improperly used heated-wire breathing circuits overheated, softened, or melted, causing diminished gas delivery, fires, and burns to patients and caregivers.[4]

Reporting does make a difference. Unfortunately, most practitioners do not realize this and fail to notify the FDA of medical-product-associated problems that they encounter. Through MEDWATCH the FDA hopes to change this behavior and to make reporting on adverse events and product problems a fundamental part of the medical culture.

What To Report

The Agency does not want and cannot handle reports on every adverse event observed. The FDA would like to know about those *serious* events in which a device or medication (either prescription or over-the-counter) was associated with death, a life-threatening condition, initial or prolonged hospitalization, disability, congenital anomaly, or when medical or surgical intervention was required to prevent permanent disability or damage.

It is not necessary to prove causality. Simple suspicion of a possible association is sufficient reason to notify the FDA.

The FDA also is interested in learning about product quality problems such as inaccurate or unreadable product labeling, packaging or product mix-up, contamination or stability problems, and particulate matter in injectable products. Malfunctioning devices that, if used, are likely to result in death or serious injury to patients should be reported as well.

How To Report

MEDWATCH has made reporting easier. Any adverse event or product problem with any FDA-regulated medical product can be reported on the new postage-paid MEDWATCH form. The only exceptions are vaccines, which, because of different reporting requirements, will continue to be reported to the Vaccine Adverse Events Reporting System (VAERS), a joint FDA/CDC project, at 1-800-822-7967.

The MEDWATCH form can be found on Pages 1079-1080 of this issue of RESPIRATORY CARE and also in numerous other publications, including the *FDA Medical Bulletin* and the 1994 *Physicians' Desk Reference.* In addition, a form can be obtained from the FDA via fax or mail by calling the 24 hour, 7-day-a-week, toll-free number 1-800-FDA-1088. This number can also be used to request a copy of the *FDA Desk Guide for Adverse Event Product Problem Reporting,* which includes instructions for completing the form.

Completed reports may be mailed to the FDA using the postage-paid form or faxed using 1-800-FDA-0178. Reports may also be submitted via computer modem by dialing 1-800-FDA-7737 and responding to a series of prompted questions.

Reports from health professionals are often the first signal the FDA has that a problem exists. Input from physicians and other practitioners can and does directly affect patient care in this country. Working in partnership through the MEDWATCH program, the FDA and the medical community along with industry can help ensure an active effective national postmarketing surveillance system.

Dianne L Kennedy MPH RPh
Director, MEDWATCH
Food and Drug Administration
Rockville, Maryland

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MEDWATCH

THE FDA MEDICAL PRODUCTS REPORTING PROGRAM

For VOLUNTARY reporting by health professionals of adverse events and product problems

Form Approved: OMB No. 0910-0291 Expires: 12/31/94
See OMB statement on reverse

FDA Use Only (RESP CARE)

Triage unit sequence #

Page ___ of ___

A. Patient information

1. Patient identifier
In confidence

2. Age at time of event:
or
Date of birth:

3. Sex
☐ female
☐ male

4. Weight
___ lbs
or
___ kgs

B. Adverse event or product problem

1. ☐ Adverse event and/or ☐ Product problem (e.g., defects/malfunctions)

2. Outcomes attributed to adverse event (check all that apply)
☐ death ___ (mo/day/yr)
☐ life-threatening
☐ hospitalization – initial or prolonged
☐ disability
☐ congenital anomaly
☐ required intervention to prevent permanent impairment/damage
☐ other:

3. Date of event (mo/day/yr)

4. Date of this report (mo/day/yr)

5. Describe event or problem

6. Relevant tests/laboratory data, including dates

7. Other relevant history, including preexisting medical conditions (e.g., allergies, race, pregnancy, smoking and alcohol use, hepatic/renal dysfunction, etc.)

C. Suspect medication(s)

1. Name (give labeled strength & mfr/labeler, if known)
#1
#2

2. Dose, frequency & route used
#1
#2

3. Therapy dates (if unknown, give duration) from/to (or best estimate)
#1
#2

4. Diagnosis for use (indication)
#1
#2

5. Event abated after use stopped or dose reduced
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

6. Lot # (if known)
#1
#2

7. Exp. date (if known)
#1
#2

8. Event reappeared after reintroduction
#1 ☐ yes ☐ no ☐ doesn't apply
#2 ☐ yes ☐ no ☐ doesn't apply

9. NDC # (for product problems only)
– –

10. Concomitant medical products and therapy dates (exclude treatment of event)

D. Suspect medical device

1. Brand name

2. Type of device

3. Manufacturer name & address

4. Operator of device
☐ health professional
☐ lay user/patient
☐ other:

5. Expiration date (mo/day/yr)

6.
model #
catalog #
serial #
lot #
other #

7. If implanted, give date (mo/day/yr)

8. If explanted, give date (mo/day/yr)

9. Device available for evaluation? (Do not send to FDA)
☐ yes ☐ no ☐ returned to manufacturer on ___ (mo/day/yr)

10. Concomitant medical products and therapy dates (exclude treatment of event)

E. Reporter (see confidentiality section on back)

1. Name, address & phone #

2. Health professional?
☐ yes ☐ no

3. Occupation

4. Also reported to
☐ manufacturer
☐ user facility
☐ distributor

5. If you do NOT want your identity disclosed to the manufacturer, place an " X " in this box. ☐

FDA

Mail to: MEDWATCH
5600 Fishers Lane
Rockville, MD 20852-9787

or FAX to:
1-800-FDA-0178

FDA Form 3500 (6/93)

Submission of a report does not constitute an admission that medical personnel or the product caused or contributed to the event.

ADVICE ABOUT VOLUNTARY REPORTING

Report experiences with:

- medications (drugs or biologics)
- medical devices (including in-vitro diagnostics)
- special nutritional products (dietary supplements, medical foods, infant formulas)
- other products regulated by FDA

Report SERIOUS adverse events. An event is serious when the patient outcome is:

- death
- life-threatening (real risk of dying)
- hospitalization (initial or prolonged)
- disability (significant, persistent or permanent)
- congenital anomaly
- required intervention to prevent permanent impairment or damage

Report even if:

- you're not certain the product caused the event
- you don't have all the details

Report product problems – quality, performance or safety concerns such as:

- suspected contamination
- questionable stability
- defective components
- poor packaging or labeling

How to report:

- just fill in the sections that apply to your report
- use section C for all products except medical devices
- attach additional blank pages if needed
- use a separate form for each patient
- report either to FDA or the manufacturer (or both)

Important numbers:

- 1-800-FDA-0178 to FAX report
- 1-800-FDA-7737 to report by modem
- 1-800-FDA-1088 for more information or to report quality problems
- 1-800-822-7967 for a VAERS form for vaccines

If your report involves a serious adverse event with a device and it occurred in a facility outside a doctor's office, that facility may be legally required to report to FDA and/or the manufacturer. Please notify the person in that facility who would handle such reporting.

Confidentiality: The patient's identity is held in strict confidence by FDA and protected to the fullest extent of the law. The reporter's identity may be shared with the manufacturer unless requested otherwise. However, FDA will not disclose the reporter's identity in response to a request from the public, pursuant to the Freedom of Information Act.

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Washington, DC 20201
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Preliminary Evaluation of High-Frequency Chest Compression for Secretion Clearance in Mechanically Ventilated Patients

Judy Whitman RRT, Ron Van Beusekom RRT, Suzanne Olson CRTT, Mary Worm CRTT, and Frank Indihar MD

BACKGROUND: A high-frequency chest compression (HFCC) device called the ThAIRapy System has been developed to provide secretion clearance therapy. We evaluated the safety, efficacy, and utility of the device in long-term mechanically ventilated patients. DESCRIPTION OF DEVICE: The primary components of the device are an air-pulse generator and an inflatable vest. Small gas volumes are alternately injected into and withdrawn from the vest by the air-pulse generator at a fast rate, creating an oscillatory or vibratory motion. The pulses cause the vest to inflate and deflate against the thorax of the patient. EVALUATION METHODS: We evaluated HFCC by comparing it to percussion and postural drainage therapy (P&PD); sputum production, patient comfort (PC), pulse-oximetry saturation (S_{pO_2}), heart rate (HR), and blood pressure (BP) data were collected and compared between the 2 methods. We monitored the reliability of the device and distributed a survey questionnaire to the entire respiratory therapy staff to assess utility. EVALUATION RESULTS: Nine patients completed the safety and efficacy portion of our evaluation. No significant difference was found between P&PD and HFCC in the wet weight of collected sputum, the mean change-in-percent of S_{pO_2} or the mean percent change from baseline in HR, BP, or PC. All therapists believed that the ThAIRapy System was easy to learn, and 70% considered it an acceptable alternative to P&PD. Of the staff members surveyed, 80% believed that use of the HFCC device resulted in time savings in all or most cases. There were no equipment malfunctions in 225 hours of use. CONCLUSIONS: Compared to P&PD, HFCC via the ThAIRapy System may be equally efficacious in promoting secretion clearance in long-term mechanically ventilated patients. HFCC is neither more nor less safe for patients than is P&PD using S_{pO_2}, HR, BP, and PC as outcome variables. Most of our respiratory therapists perceived HFCC as an acceptable alternative to P&PD. [Respir Care 1993;38(10):1081-1087.]

Ms Whitman is Supervisor, Mr Van Beusekom is Director, Mss Olson and Worm are Respiratory Therapists, Cardiopulmonary Services Department, and Dr Indihar is Director, Prolonged Respiratory Care Unit—Healtheast Bethesda Lutheran Hospital, St Paul, Minnesota.

The authors have no financial interest in the product described.

A version of this paper was presented by Ms Whitman at the Second International Conference on Advances in Pulmonary Rehabilitation and Management of Chronic Respiratory Failure held in Venice, Italy, November 4, 1992.

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Background

In healthy persons, secretion clearance is accomplished primarily through mucociliary action and cough.[1] Under normal conditions these mechanisms are very efficient. However, many patients suffer from impairment of the mucus transport system and are at risk for retained bronchial secretions. Inadequate secretion clearance can lead to serious complications that include infection, increased work of breathing, and hypoxemia.[2] Promoting bronchial drainage in some patient popula-

tions is a clinical challenge. For instance, mechanical ventilation often complicates our ability to provide secretion clearance therapy because some ventilated patients have weakened coughs, are difficult to properly position, and often poorly tolerate aggressive secretion clearance procedures. Over the years, several methods designed to enhance mucus clearance have been evaluated.[3-5]

Percussion and postural drainage (P&PD) is frequently employed to promote secretion clearance in patients with excessive or retained pulmonary secretions. Although P&PD appears to be appropriate for some patients,[6,7] it is a labor-intensive, and somewhat controversial, therapy.[4,8-10] Therefore, alternatives to the use of P&PD merit investigation.

Many researchers[11-19] report an increase in mucus mobilization during high-frequency oscillatory ventilation. High-frequency chest compression (HFCC) is a method for generating oscillatory airflow in the airways and thereby mobilizing pulmonary secretions. Beginning in the early 1980s, several groups evaluated the effect of HFCC on secretion clearance. In 1983, King et al[11] observed a statistically significant increase in the rate of mucus clearance after applying high-frequency pulses, via a modified blood pressure cuff, to the lower thoraxes of dogs. In 1985, Gross and associates[12] monitored regional lung clearance of a ^{99m}Tc-sulfur-colloid aerosol with a gamma camera and found significantly improved peripheral mucus clearance during HFCC. The ability of HFCC to enhance mucus mobilization may involve several mechanisms. The most likely mechanisms proposed by researchers include mucus shearing due to increased mucus-airflow interaction, enhancement of cilia beat-frequency as a result of a reflex mechanism, and facilitation of the cephalad movement of mucus by the higher expiratory flows (compared to inspiratory flows).[11]

Recently, an HFCC device called the ThAlRapy System (American Biosystems, St Paul MN) has been developed to provide secretion clearance therapy. Studies have documented increased mucus mobilization[15,16] and improved pulmonary function[17] in cystic fibrosis patients treated with the ThAlRapy System.

Based on this information, we decided to evaluate the safety, efficacy, and utility of this HFCC in long-term mechanically ventilated patients who required P&PD therapy. Our intent was to gather enough preliminary information to determine whether HFCC would be useful in this patient population.

Description and Application of Device

The primary components of the Model 102 ThAlRapy System are the air-pulse generator and an inflatable vest. The performance specifications of this product have been fully described in an earlier paper.[15] The vest, which is made of a material that does not stretch, is designed to fit over the entire thorax of a patient. Each vest has two ports, one on each side of the front panel (Fig. 1). Large-bore tubing connects the vest to the air-pulse generator. Small gas volumes are alternately injected into and withdrawn from the vest by the air-pulse generator at a very fast rate, creating an oscillatory or vibratory motion. The pulses cause the vest to inflate and deflate against the thorax of the patient. There are two operator-adjustable controls: pulse frequency and pulse pressure. Pulse frequency is variable from 5 to 25 Hz and pulse pressure from 0.35 to 0.75 psi.

Fig. 1. Secretion-clearance procedure using the ThAlRapy System performed on a mechanically ventilated patient .

One HFCC protocol,[17] described for use in cystic fibrosis patients, uses a range of frequencies. Patients are in a sitting or semi-Fowler's position and

receive treatment for 5 minutes at each frequency. At this time (1993), the medical literature does not include a description of HFCC in patients with pulmonary diseases other than cystic fibrosis. Therefore, the full range of potential applications is unknown.

The inflatable vests are applied to patients with minimal manipulation. Our entire population required a 'wardrobe' of only 6 reusable vests that ranged in size from small to extra-large. Before applying the device to patients, we considered the risk of cross-contamination. Because the vests were not used over broken or irritated skin, we classified the components of the HFCC device as noncritical.[20] Components were cleaned with a low-level hospital disinfectant before being applied to another patient.

Evaluation Methods

To evaluate the safety and efficacy of the HFCC device in long-term mechanically ventilated patients, we compared a conventional P&PD protocol to an HFCC protocol. Efficacy was evaluated by comparing the wet weight of sputum collected during therapy sessions. Safety was evaluated by monitoring patient comfort and certain physiologic variables. To evaluate the utility of the device, we developed a survey questionnaire and monitored the reliability of the device by recording malfunctions or problems that required service.

Patients

During the safety and efficacy portion of our evaluation, mechanically ventilated patients who had received artificial ventilation more than 30 days and who required external manipulation of the thorax to enhance secretion clearance (as determined by the clinical judgment of the attending physician) were recruited to participate. Protocol approval and informed consent were obtained according to institutional guidelines. Patients with a history of pneumothorax, hemoptysis, or cardiac arrhythmias within the past 30 days, and those who required a high level of ventilatory support ($\dot{V}_E$ > 15 L/min, PEEP >10 cm H_2O, F_{IO_2} > 0.50) were excluded from participation. Ten patients were enrolled in the safety and efficacy portion of our evaluation (Table 1). Patient 7 withdrew before completion of the protocol and was not included in the final analysis.

Table 1. Description of Patients at Study Entry

Patient Number	Sex/Age	Diagnosis	Days on Ventilator
1	M / 56	Multiple sclerosis	640
2	M / 71	Cerebral vascular accident	1203
3	M / 90	COPD, respiratory failure	250
4	M / 68	COPD, cerebral vascular accident	180
5	M / 76	Cerebral vascular accident	720
6	M / 40	Myotonic dystrophy	310
7	F / 62	Alcohol abuse, respiratory failure	260
8	F / 74	Emphysema, respiratory failure	90
9	F / 70	Amyotrophic lateral sclerosis	250
10	M / 72	Amyotrophic lateral sclerosis	480

Study Design

A crossover design, with each patient enrolled for a period of 4 days, was used for this study. Randomization of subjects into the 2 therapy groups was accomplished by using a random, permuted-block design with crossover that allowed for 2 consecutive days of each form of therapy for each subject. In each patient, both HFCC and P&PD were performed according to the same schedule, either 3 or 4 times/day. Five trained respiratory therapists performed all therapies.

P&PD Protocol

Percussion was applied for 2 minutes over each of 5 regions: anterior segments of both upper lobes, right-upper-lobe posterior segment, right-lower-lobe lateral segment, left-upper-lobe posterior segment, and left-lower-lobe lateral segment. Patients were positioned 15° Trendelenburg for drainage of lower lobes.

HFCC Protocol

HFCC was administered at 8 Hz and 16 Hz, each for 5 minutes, according to the manufacturer's

recommendations. Therapy was performed with patients sitting or in the semi-Fowler's position and proceeded from the lowest to the highest frequency. HFCC was applied during both inhalation and exhalation throughout the entire 10-minute therapy session.

Data Collection

The total time of active treatment was the same for both secretion clearance techniques. Patients were suctioned prior to the start of all therapies. This pretherapy sputum sample was not measured or included in the total amount of sputum cleared during the therapy. Sputum was collected during the therapy session, immediately at the end of the therapy, and 30 minutes after therapy completion. Sputum samples were collected in 40-mL specimen traps that were weighed prior to sputum collection. The wet-weight determination of the sputum was performed immediately after obtaining the last therapy-session sample. Our primary outcome variables for the assessment of safety were arterial oxygen saturation by pulse oximeter (S_{pO_2}), heart rate (HR), blood pressure (BP), patient comfort (PC), and the wet weight of sputum cleared during therapy sessions.

HR and S_{pO_2} were measured with a pulse oximeter (Model N-200, Nellcor Inc, Hayward CA) before, after 5 minutes of therapy (treatment midpoint), and after each therapy. BP was measured before, during, and after each therapy with a noninvasive blood pressure monitor (Model 1846, Dinamap, Critikon Inc, Tampa FL). Patient comfort was assessed before, during, and after each therapy by having the patients indicate their comfort level on a visual analog scale. We asked the patients to point to a number corresponding to their perceived level of comfort, on a scale of 1 (no discomfort) to 10 (extreme discomfort). Their responses were recorded for PC analysis. Our analysis of HR, S_{pO_2}, BP, and PC consisted of comparing the pretherapy measure to the during-therapy measure and determining the average change in S_{pO_2} % and % change from baseline for HR, BP, and PC. These data were positive when the values for the HFCC group were higher than those for the P&PD group. Sputum production was analyzed by comparing the mean weight of the sputum collected with each treatment method. All comparisons were made using the Wilcoxon signed-rank test for matched pairs.

To evaluate the utility of HFCC, we developed a survey questionnaire that used a 5-point Likert scale (Fig. 2) to collect information regarding ease of learning, therapist acceptance, and perceived impact on productivity. The questionnaire was distributed to our entire staff of 20 respiratory therapists approximately 3 months after the patient portion of the study had been completed. We collected information from the entire population of respiratory therapists, and summarized the results of the survey using simple descriptive statistics. To quantify the number of malfunctions or equipment problems, we divided the hours of use (obtained from a hour meter on the device) by the number of malfunctions or problems to arrive at problems per hour of us.

User Survey

1. Approximately how many therapies have you performed with the ThAIRapy System?
   ☐ < 25 ☐ 25-50 ☐ > 50
2. The ThAIRapy system is easy to learn?
   ☐ in all cases ☐ in most cases
   ☐ in some cases ☐ in no cases ☐ N/A
3. The ThAIRapy system is an acceptable alternative to CPT?
   ☐ in all cases ☐ in most cases
   ☐ in some cases ☐ in no cases ☐ N/A
4. The ThAIRapy system results in time savings?
   ☐ in all cases ☐ in most cases
   ☐ in some cases ☐ in no cases ☐ N/A

Fig. 2. Survey questionnaire administered to respiratory care staff. Responses were used to evaluate HFCC utility.

Evaluation Results

For the 9 patients who completed the study, 88 treatments were performed. The mean (SD) weight of sputum specimens collected with each therapy was HFCC 3.60 (3.4) g and P&PD 3.39 (2.1) g. These data for each patient are shown in Figure 3. No significant difference in the wet weight of col-

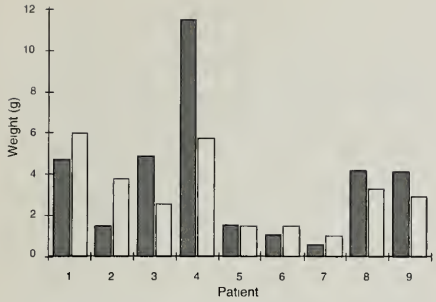

Fig. 3. Mean wet weight of collected pulmonary secretions per patient, by treatment method: HFCC = ■, P&PD = □.

lected sputum was found between the two therapy techniques (p = 0.77). The mean change in S_{pO_2} % and mean % change from the pretherapy measures to the during-therapy measures for BP, HR, and PC are reported in Table 2. When HFCC was compared to P&PD, no significant difference was found in the mean % change from baseline for any variable. Due to communication difficulties, 3 of the 9 patients were judged unable to provide reliable comfort-level data and our results are based only on the remaining 6 patients (Table 2). Questionnaires were distributed to the entire 20-member respiratory therapy staff. All questionnaires (100%) were properly completed, returned to the study coordinator, and tabulated for final results. We found that 19 of 20 of the respiratory therapy staff members had performed at least 25 treatments using HFCC. Our therapists believed that HFCC was easy to learn to use, and 70% considered it an acceptable alternative to P&PD in most circumstances. Eighty percent of the staff felt that use of the HFCC device often or always resulted in time savings. During a 6-month period that represented 225 hours of use, there were no equipment malfunctions or problems that required service.

Table 2. Summary of Results Describing the Mean Change in Safety-Assessment Measures between Pretherapy and During-Therapy Values

Measure	Technique		p
	HFCC	P&PD	
Blood Pressure (% change)	0.02% (7.2%)*	1.06% (6.2%)	0.59
Heart Rate (% change)	1.71% (2.5%)	4.21% (3.2%)	0.09
S_{pO_2} (change in %)	0.90% (0.9%)	0.41% (1.81%)	0.59
Comfort Level (% change)	135.0% (79.2%)†	151.2% (170.1%)	0.92

*Results are mean (SD).
†Based on 6 patients; higher score = greater discomfort.

Discussion

Providing secretion clearance therapy in many patient populations is a clinical challenge. We undertook an evaluation to explore the safety, efficacy, and utility of the ThAIRapy System in stable mechanically ventilated patients who required assistance with pulmonary-secretion clearance. We recorded and compared S_{pO_2}, HR, BP, PC, and the wet weight of collected sputum in 9 chronically ventilated patients. In this preliminary evaluation, we found HFCC via the ThAIRapy System to be as effective as P&PD for secretion mobilization. Additionally, HFCC was found to be a safe method of secretion clearance when compared to a conventional P&PD protocol, and the results of our survey questionnaire point to a high level of HFCC utility from the perspective of front-line respiratory therapists.

The ability of oscillatory airflow to enhance secretion mobilization is described in the literature.[11,19] However, we did not observe a statistically significant increase in sputum volume during HFCC when compared to P&PD. Perhaps a larger group of subjects would have evoked significance. Our results may be due to some inadequacy of the measure, or it may be because many of our patients suffer from some form of neuromuscular weakness and possess weak or nonexistent coughs—consequently, we may not have captured all the secretions loosened during therapy. Given our data on sputum production, HFCC compared well to a rigorous P&PD procedure that included Trendelenburg positioning and percussion to all lobes.

In most patients, both P&PD and HFCC were well tolerated as shown by the similar changes in S_{pO_2}, BP, HR, and PC. However, on two occasions, one patient in the P&PD group experienced an S_{pO_2} drop of 6% while in the Trendelenberg position; the saturation decrease was presumably related to

the position because return to normal saturation occurred when the patient was returned to an upright position. Interestingly, the largest S_{pO_2} drop during HFCC therapy for the same patient was only 2%. Therefore, the ability to effectively promote secretion clearance without head-down positioning may be an advantage of HFCC over P&PD.

It is important that the equipment we apply to patients be not only safe and efficacious but also useful and reliable. Medical devices should possess attributes that facilitate the efficient delivery of patient care. With an average of 9,166 patient-ventilator days and 7,200 P&PD therapies per year, heavy demands are placed on our department. As we face the challenges of expanding roles, staff shortages, and cost containment, it is important to continually evaluate alternative methods of delivering patient care. The availability of various methodologic options gives therapists the ability to choose and apply the most appropriate form of therapy. HFCC was well accepted by our respiratory therapy staff. A high percentage of therapists (80%) believed that there were time savings associated with HFCC therapy in all or most cases. Objective time studies are required to better understand issues related to therapist productivity and the use of HFCC. We found no objective evidence that the HFCC device is superior or inferior to P&PD. However, subjectively, HFCC appears to be safe and well tolerated by the patients, well received by the caregivers, and somewhat less labor-intensive than P&PD.

We did not perform a formal cost-analysis; however, the time saved by the use of HFCC (if, in fact, time is saved) must be balanced against the cost of the ThAIRapy System (Rent $500/mo), disposable vest and tubing ($98.50/patient), and subsequent maintenance costs. Additional questions beyond the scope of this evaluation remain:

- What are the benefits of HFCC from a patient outcome perspective?
- Are there long-term economic advantages of HFFC?
- Would postural drainage enhance the effect of HFFC therapy?

With our preliminary safety, efficacy, and utility questions answered, we plan to conduct additional research to explore these issues.

Conclusions

In this evaluation, we did not find HFCC via the ThAIRapy system to be more or less effective for secretion clearance than P&PD; there was no difference in our measurements of safety (S_{pO_2}, HR, BP, and PC) between therapy methods; and secretion clearance using an HFCC device received a high utility rating from our respiratory therapy staff. Although much more research is required, the results of our evaluation lead us to believe that HFCC is a viable alternative to P&PD for secretion clearance in chronically ventilated patients.

ACKNOWLEDGMENTS

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Cyanosis and Sulfhemoglobinemia: A Case Report

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Introduction

Sulfhemoglobinemia is an uncommon disorder that is often mistaken for methemoglobinemia. Both of these disorders cause cyanosis and result from alteration of the heme moiety by certain drugs and chemicals, usually when such substances are taken in excessive amounts.[1-4] Although sulfhemoglobinemia and methemoglobinemia have similar spectral characteristics, important clinical and laboratory differences exist between the two abnormal pigments. I describe a patient who developed sulfhemoglobinemia only a few days after beginning to take therapeutic doses of a drug known to cause methemoglobinemia.

Case Summary

An 80-year-old White woman was referred to the Pulmonary Medicine Department by her dermatologist who had requested arterial blood gas analysis because he had detected cyanosis of her lips and fingertips. The patient recalled that she had noticed the bluish color of her skin but was not sufficiently alarmed to seek medical attention. Arterial blood drawn while the patient was breathing room air was chocolate colored. Analysis revealed pH 7.44, P_{aCO_2} 41 torr [5.5 kPa], and P_{aO_2} 76 torr [10.1 kPa]. Subsequent analysis of a blood sample by CO-oximeter (IL-282, Instrumentation Laboratory, Lexington MA) revealed a methemoglobin level of 42%. Because of the patient's age and a remote history of angina, she was admitted to the hospital for further evaluation of her cyanosis.

Prior to her admission, the patient was in her usual state of fair health, with mild diabetes and chronic urinary tract infections due to an atonic bladder that required self-catheterization 2-3 times daily. There was no history of any cardiac or pulmonary disease, and she was asymptomatic except for mild exertional dyspnea.

On physical examination the patient was cachectic, and her hue was violaceous and slightly yellow in appearance. Marked circumoral cyanosis was present, and her nail beds had a slate-gray appearance. Her regular medications included alprazolam (Xanax) 0.75 mg daily, nalidixic acid (Negram) 1 g daily, and metaprolol tartrate (Lopressor) 50 mg daily. Five days prior to her admission, phenazopyridine hydrochloride (Pyridium) had been prescribed for urinary tract discomfort, and she began taking 200 mg daily.

Her vital signs were normal, and the lungs were clear to auscultation and percussion. Her cardiovascular examination was unremarkable with no murmur heard. The chest roentgenogram revealed no active disease. Laboratory studies revealed a hemoglobin of 12.2 g/dL with normal red cell morphology and indexes. The white blood cell count was 9,900/mm^3 with a normal differential. The blood urea nitrogen level was 13 mg/dL with a creatinine value of 0.7 mg/dL. The glucose-6-phosphate dehydrogenase (G6PD) level was within normal limits. Hemoglobin M was not detected. The arterial blood was brown.

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A preliminary diagnosis of methemoglobinemia was made, and the phenazopyridine was stopped. Methylene blue (1 mg/kg) was given intravenously over a 5-minute period. One hour later her color was unchanged and a repeat methemoglobin level was 40%. A second dose of methylene blue was administered with no observable diminution in her cyanosis. CO-oximeter analysis revealed a methemoglobin level of 39.3%. Because the patient's cyanosis and methemoglobin levels failed to respond to methylene blue administration, a sample of her blood was sent to a university facility for further evaluation. Detailed spectrophotometric analysis revealed a sulfhemoglobin level of 18.3% and an absence of methemoglobin. Five months after discharge, spectrophotometric analysis was repeated and revealed no evidence of sulfhemoglobin. Her cyanosis had disappeared.

Discussion

Cyanosis is most commonly due to an increased amount of reduced hemoglobin in circulating blood caused by either an anatomic cardiac right-to-left shunt or a disturbed ventilation/perfusion relationship in the lung.[5,6] In the evaluation of a cyanotic patient with no evidence of cardiopulmonary disease, the differential diagnosis must include the dyshemoglobinemias, specifically methemoglobinemia and sulfhemoglobinemia. In patients with either of these abnormal pigments, the slate-gray color of the skin is not due to any cardiopulmonary deficiency but is rather the result of the spectral characteristics of the abnormal pigments. However, elevated levels of the pigments can affect oxygenation by their inability to bind oxygen and by their effect on the oxygen dissociation curve. Sulfhemoglobin is an incompletely characterized hemoglobin derivative. While the precise structure of the pigment is uncertain, isoelectric-focusing techniques have determined that the sulfhemoglobin molecule has a sulfur atom incorporated into the porphyrin ring.[7] Sulfhemoglobin has a characteristic absorption peak of 618 nm, which is not abolished with the addition of cyanide. Its name is derived from its in-vitro production by the chemical reaction of hydrogen sulfide with hemoglobin. The mechanism for the production of sulfhemoglobin in vivo is not well understood.[7-9]

Sulfhemoglobinemia is most often seen in patients exposed to certain chemicals or those taking certain drugs.[1,3,7] Many of the substances known to cause sulfhemoglobinemia can also cause methemoglobinemia. Finch listed 30 agents known to cause either sulfhemoglobinemia or methemoglobinemia.[5] The most common offenders included nitrate compounds, sulfonamides, and aniline derivatives. Phenazopyridine hydrochloride, an azo dye and the offender in this case, is a commonly used urinary tract analgesic. Phenazopyridine's side effects include hemolysis, renal failure, yellow skin pigmentation, and methemoglobinemia.[10] These side effects are most often seen in association with overdoses.[2,11,12] Phenazopyridine is an indirect oxidant and does not produce sulfhemoglobin (or methemoglobin) in vitro.[13] Its oxidative toxicity is most likely a result of its conversion to some intermediate forms that are direct oxidants. A number of cases of phenazopyridine-induced methemoglobinemia have been reported in the literature.[2,11,12,14-16] Only rarely has phenazopyridine been associated with sulfhemoglobinemia.[6,17] It is interesting to note that Jeffery and co-workers described two cases of methemoglobinemia secondary to phenazopyridine ingestion that did not respond to methylene blue administration.[18] The clinical presentation of these cases was more consistent with sulfhemoglobinemia. Indeed, there are a number of reports in the medical literature in which sulfhemoglobinemia was mistaken for methemoglobinemia.[3,7,17,19] Despite a review of 62 cases from the Mayo Clinic that concluded that sulfhemoglobinemia is more common than methemoglobinemia,[4] the former is treated as an afterthought to the latter in the medical literature.[20]

Park, in her thorough review of sulfhemoglobin, expresses her belief that sulfhemoglobinemia is often under diagnosed, most likely because of inadequate laboratory documentation.[7] The laboratory documentation of sulfhemoglobinemia requires special equipment that was not available to us. The patient in this report was diagnosed as having methemoglobinemia based on her physical examination, her history of taking a medication known to cause methemoglobinemia, and a CO-oximetry analysis that revealed an abnormal methemoglobin level.

The IL-282 CO-oximeter is an automated spectrophotometer that uses a 4-wavelength method to

detect the optical absorbance of hemoglobin fractions in whole blood. The four wavelengths measured are 530.0, 585.2, 594.5, and 626.6 nm. This methodology allows for the determination of oxyhemoglobin, deoxyhemoglobin, carboyhemoglobin, and methemoglobin. Unfortunately, the IL-282 cannot distinguish between methemoglobin and sulfhemoglobin. Because sulfhemoglobin's absorbance peak of 618 nm is close to methemoglobin's peak of 630 nm, the sulfhemoglobin exhibits spectral interference with the methodology of the IL-282. The presence of sulfhemoglobin results in an abnormally high methemoglobin value. The manufacturer acknowledges this problem.[21] Halvorsen & Dull[17] and Kelner and Bailey[19] reported similar experiences with the IL-282 with sulfhemoglobin mismeasured as methemoglobin.

Although sulfhemoglobinemia is often mistaken for methemoglobinemia, the differences between the two abnormal hemoglobin derivatives are not trivial. Table I lists several characteristics that allow differentiation between sulfhemoglobinemia and methemoglobinemia. As little as 0.5 g/dL of sulfhemoglobin produces readily detectable cyanosis, whereas 1.5 g/dL of methemoglobin and 5.0 g/dL of reduced hemoglobin are necessary to produce similar degrees of cyanosis.

Table 1. Clinical and Laboratory Differences between Methemoglobinemia and Sulfhemoglobinemia

Characteristic	Methemoglobin	Sulfhemoglobin
Oxygen binding	No	No
O_2 curve shift	Left	Right
Detectable cyanosis	1.5 g/dL	0.5 g/dL
Toxic level	> 30%	> 60%
Reversible	Yes	No
Duration	Drug dependent	Life of RBC*
Spectrophotometry peak	630 nm	618 nm
CO-oximetry detectable	Yes	Some systems

*Red blood cell.

Neither sulfhemoglobin nor methemoglobin can bind with oxygen. Their effect on the oxygen transport system is also determined by how the abnormal pigments affect neighboring unmodified hemes. Methemoglobin's structural conformation causes adjacent unmodified hemes to have a greater affinity for oxygen and therefore a leftward shift of the oxygen dissociation curve. Sulfhemoglobin's unliganded conformation, on the other hand, causes neighboring normal hemes to have a reduced affinity for oxygen and a rightward shift of the curve, resulting in enhanced oxygen delivery to the periphery. This difference most likely explains why patients with sulfhemoglobinemia are generally less toxic than those with methemoglobinemia despite the fact that these patients look bluer than those with methemoglobinemia. Sulfhemoglobin levels as high as 60% of the total pigment rarely cause clinically important signs or symptoms. Similar levels of methemoglobin may be associated with vascular collapse, coma, and death.[8]

It is fortunate that sulfhemoglobin produces a rightward shift in the oxygen dissociation curve because there is no antidote for sulfhemoglobinemia. Unlike methemoglobin, which can be reduced to normal hemoglobin by the administration of methylene blue, no such conversion process exists for sulfhemoglobin. The pigment lasts the life of the erythrocyte, approximately 120 days. In fact, sulfhemoglobin-containing red cells have been used to determine erythrocyte lifespan.[9] The treatment of sulfhemoglobinemia is withdrawal of the offending agent. Exchange transfusion is rarely needed because of the low toxicity of the pigment.

Sulfhemoglobinemia is essentially a benign disorder, although the cyanosis can be cosmetically distressing. The present report describes a patient with normal renal and hematologic function who developed sulfhemoglobinemia soon after beginning to take therapeutic doses of a medication known to cause methemoglobinemia when taken in excessive amounts. It is unclear why this patient developed sulfhemoglobinemia rather than methemoglobinemia, although the lists of causative agents for both these disorders overlap. The origin of the sulfur atom necessary to produce sulfhemoglobin is also unclear because the patient was not taking any sulfur-containing drugs or was not exposed to any sulfur-containing chemicals. Halvorsen & Dull were also unable to discover the origin of the sulfur atom in their report of a case of phenazopyridine-induced sulfhemoglobinemia.[17] Chronic constipation with the production of excess hydrogen sulfide in the gut has been postulated as the source of the sulfur atom, but this has never been proven.[20] Whatever the etiology, the con-

centration of phenazopyridine in this patient presumably was sufficient to overwhelm the counteroxidant protective mechanisms within the erythrocyte and resulted in the development of sulfhemoglobinemia.

Despite the unknown etiology of this disorder, what is certain is that careful laboratory documentation is necessary to differentiate between methemoglobin and sulfhemoglobin. Not all commercially available CO-oximeter systems are able to differentiate between these two pigments. Operators of such instrumentation must take care to ascertain the limitations of CO-oximeter systems when analyzing blood samples for the presence of abnormal hemoglobin.

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Noninvasive Bi-Level Positive Pressure Ventilation: Management of Two Pediatric Patients

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Introduction

Hypoxemia can occur when ventilation-perfusion mismatch results from a reduction in a patient's functional residual capacity (FRC). This can be observed in patients with adult respiratory distress syndrome, pulmonary edema, and atelectasis. Respiratory management in this group of patients is generally directed toward maneuvers that increase lung volume. This frequently includes endotracheal intubation and the institution of conventional mechanical ventilation including positive end-expiratory pressure (PEEP) by volume- or pressure-limited ventilator. We describe our experience with two pediatric patients who developed atelectasis and pulmonary edema and were successfully managed noninvasively with a bi-level positive airway pressure device—a flow-cycled, pressure-limited ventilator, coupled with a nasal mask.

Device Description

The bi-level positive airway pressure device that we used (BiPAP®, Respironics Inc, Murrysville PA) provides pressure-limited ventilation with PEEP, or expiratory positive airway pressure (EPAP), in response to the patient's inspiratory effort. The device provides EPAP ranging from 2-20 cm H_2O [0.2-2.0 kPa], and inspiratory positive airway pressure (IPAP) ranging from 2-25 cm H_2O [0.2-2.5 kPa]. Electronic pressure controls also allow selection of cycling frequency between IPAP and EPAP (from 6-30 cycles/min) and the % IPAP, or proportion of each cycle spent at IPAP, ranging from 10-90%.

The system[1] incorporates a contoured nasal mask secured in place with a headband and connected to the ventilator. The system is interactive with patient breathing (ie, it responds to patient inspiratory effort) through the use of a flow transducer. At the initiation of inspiration, a change in the level of circuit flow causes the device to impose a preset IPAP. As inspiratory flow decreases toward the end of the inspiratory cycle, the pressure-supported breath is terminated in synchrony with the patient's breathing pattern. The pressure in the circuit then drops to a preset EPAP allowing the patient to exhale. The unit cycles between the set EPAP and IPAP levels in response to patient triggering, requiring only a 40 mL/s inspiratory flow to initiate ventilatory support and capable of delivering peak flows of 180 L/min. In the spontaneous mode, patients control their own respiratory frequency, inspiratory flow, and expiratory time, while the device augments the patient's tidal volume.

Case Presentation 1

TG was a 12-year-old boy with Down's Syndrome, admitted following a 1-week history of gastroenteritis. His past medical history was remarkable for episodes of absence seizures (petite mal) diagnosed at 6 months of age, insulin-dependent diabetes mellitus presenting at 1 year of age, and

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hypothyroidism. The patient had experienced frequent hospitalizations for croup, intractable diabetes, congestive heart failure, and pulmonary edema.

When the boy was admitted to the University of Michigan Pediatric Intensive Care Unit (PICU), examination revealed a child with the stigmata of Down's Syndrome who was small for his age. He was pale, afebrile, and in respiratory distress. The physical examination revealed bilateral periorbital edema and diminished bilateral coarse breath sounds on auscultation. The liver was palpable 5 cm below the right costal margin. Cardiovascular examination was normal, and an echocardiogram demonstrated normal cardiac anatomy, without evidence of pulmonary artery hypertension.

Important laboratory findings included albumin 2.2 g/dL (normal range 3.5-4.5 g/dL), serum creatinine 2.0 mg/dL (0.4-0.6 mg/dL), potassium 5.6 mEq/L (3.6-5.5 mEq/L), and blood urea nitrogen 16 mg/dL (5-20 mg/dL). Urinalysis revealed 1+ proteinuria with a specific gravity of 1.030. The chest radiograph was consistent with pulmonary edema.

Endotracheal intubation and mechanical ventilation were instituted with moderate levels of PEEP (6-8 cm H_2O) until Day 17 when he was extubated. On Day 18, his clinical condition deteriorated. A chest radiograph demonstrated a right lower-lobe infiltrate with loss of lung volume in the right lower lung (Fig. 1A). Arterial blood gas (ABG) values on a fractional concentration of delivered oxygen (F_{DO_2}) of 0.98 by face mask showed a pH 7.44, P_{aCO_2} 43 torr, P_{aO_2} 46 torr, and arterial oxyhemoglobin saturation (S_{aO_2}) 83%. The prospect of a protracted illness and prolonged course of endotracheal intubation prompted the trial of noninvasive (nasal mask) bi-level positive airway pressure ventilation in the spontaneous mode. The initial settings were 12 cm H_2O IPAP and 6 cm H_2O EPAP, giving a 'pressure boost' of 6 cm H_2O (IPAP – EPAP). Marked clinical improvements in the patient's condition were observed (Fig. 1B). ABG values were pH 7.41, P_{aCO_2} 45 torr, P_{aO_2} 68 torr, and S_{aO_2} 93%. The airway pressures and oxygen concentrations were gradually reduced (F_{DO_2} = 0.35) with S_{aO_2} maintained at > 90%. The oxygen concentration provided by the device (F_{DO_2}) was then reduced to 0.21, and on Day 21 the patient was placed on oxygen by nasal cannula at 2 L/min. ABG values were pH 7.42, P_{aCO_2} 45 torr, P_{aO_2} 65 torr, and S_{aO_2} 91%.

Fig. 1A. Chest radiograph (anteroposterior, or A-P, view) of 12-year-old boy (Case 1) demonstrates a right lower-lobe infiltrate and diminished lung volumes consistent with atelectasis.

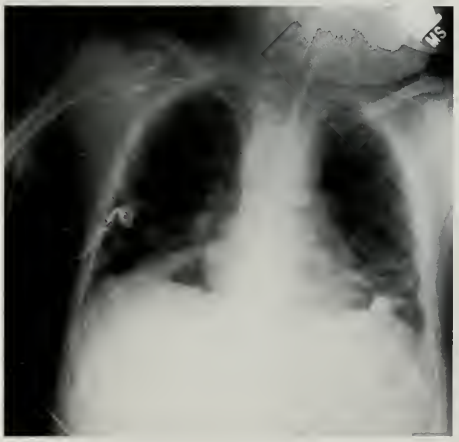

Fig. 1B. Chest radiograph of the same 12-year-old boy on Day 2 after institution of bi-level positive airway pressure. Findings are consistent with lung re-expansion.

On Day 25, oxygen requirements again increased. The chest radiograph revealed diffuse, fluffy interstitial infiltrates (Fig. 2A). Hemo-

Fig. 2A. Chest radiograph (A-P view) of same 12-year-old boy taken on Day 25 reveals diffuse interstitial markings. Oxygen requirements had increased after 4 days of spontaneous breathing without ventilatory support.

Fig. 3A. Chest radiograph (A-P view) of the same 12-year-old boy taken on Day 42 after a period of spontaneous breathing reveals diminished lung volume of the left lower lung.

Fig. 2B. Repeat radiograph of same 12-year-old boy taken after 24 hours on bi-level positive airway pressure ventilation and the administration of diuretics. Improvement can be seen.

Fig. 3B. Chest radiograph of the same 12-year-old boy on Day 44 after 2 days of bi-level positive airway pressure ventilation. Re-expansion of the lung can be seen.

dynamic measurements indicated a pulmonary capillary wedge pressure of 14 mm Hg with a cardiac index of 4.5 $L \cdot min^{-1} \cdot m^{-2}$. Noninvasive bi-level positive-pressure ventilation with supplemental oxygen was reinstituted for 3 more days and diuretics were administered. The patient's condition improved, and a chest radiograph taken after 24 hours of noninvasive positive-pressure ventilation showed remarkable improvement (Fig. 2B). The patient's respiratory status continued to improve during the next week. On Day 42, during unassisted spontaneous breathing, the patient experi-

enced another episode of respiratory distress accompanied by tachypnea, tachycardia, and restlessness. A chest radiograph revealed atelectasis of the lower left lung (Fig. 3A). Noninvasive ventilation was reinstituted, and ABG values on F_{DO_2} 0.55 were pH 7.44, P_{aCO_2} 24 torr, P_{aO_2} 38 torr, and S_{aO_2} 76%. The IPAP was increased from 18 to 20 cm H_2O, and the EPAP was maintained at 8 cm H_2O. ABG values were pH 7.43, P_{aCO_2} 33 torr, P_{aO_2} 96 torr, and S_{aO_2} 98%. After 2 more days of noninvasive positive-pressure ventilation, the patient was weaned to a face mask (F_{DO_2} = 0.28). The chest radiograph on Day 44 showed marked improvement (Fig. 3B). The patient's pulmonary condition remained stable thereafter; however, he later succumbed to postoperative complications.

Case Presentation 2

RB was a 12-year-old boy with acute T-cell lymphoblastic leukemia who presented to the Emergency Department of Mott Children's Hospital at the University of Michigan with a 2-day history of nausea, vomiting, diarrhea, and fever. He had recently completed a course of chemotherapy that included cytosine-arabinoside and L-aspariginase. On admission, the child required vigorous volume resuscitation and was placed on broad spectrum antimicrobial drugs. He was subsequently transferred to the PICU on Day 7.

Physical evaluation was remarkable for acute respiratory distress and fever. Vital signs included a temperature of 39.3°C, pulse rate 150/min, and respiratory rate of 50/min. Blood pressure with a dopamine infusion (5 $\mu g \cdot kg^{-1} \cdot min^{-1}$) was 130/80 mm Hg. Breath sounds were diminished bilaterally, especially over the right lower-lung field. The chest radiograph was remarkable for a right lower-lobe infiltrate with diminished lung volumes and a diffuse, fluffy infiltrate bilaterally. His S_{aO_2} was 80% while breathing 60% oxygen.

Laboratory results included a potassium of 2.9 mEq/L (normal values 3.6-5.5 mEq/L), bicarbonate of 15 mEq/L (22-25 mEq/L), blood urea nitrogen of 13 mg/dL (5-20 mg/dL), and creatinine of 1.2 mg/dL (0.4-0.6 mg/dL). Serum albumin was 2.5 g/dL (3.5-4.5 g/dL). The absolute neutrophil count was 200/mm^3 (2.3-7.23/mm^3). A blood culture grew *Streptococcus mitis*.

S_{aO_2} was 64% with a P_{aO_2} of 32 torr while on a face mask at F_{DO_2} = 0.60. The patient was begun on noninvasive positive-pressure ventilation due to increasing oxygen requirements and tachypnea. The initial settings were F_{DO_2} 0.28, IPAP 14 cm H_2O, and EPAP 6 cm H_2O, giving a pressure boost of 8 cm H_2O. ABG values were pH of 7.48, P_{aCO_2} 24 torr, P_{aO_2} 68 torr, and S_{aO_2} 95%. On Day 8, fiberoptic bronchoscopy was performed. Bronchial washings gave no evidence of bacterial, fungal, or parasitic infection. Clinical examination was remarkable for basal crackles, diffuse wheezing, and a weight increase of 2 kg. Chest radiograph revealed increased alveolar markings. A cardiovascular examination including echocardiogram was normal. Furosemide and chlorothiazide were given intravenously. The IPAP was increased from 14 to 16 cm H_2O, and the EPAP from 6 to 8 cm H_2O (pressure boost of 8 cm H_2O). ABG values with F_{DO_2} = 0.45 were pH 7.49, P_{aCO_2} 32 torr, P_{aO_2} 79 torr, and S_{aO_2} 96%.

On Day 10, granulocyte colony stimulating factor was administered. The patient was febrile (temperature 40°C) with a respiratory rate of 37/min. The positive-pressure nasal mask was temporarily removed and oxyhemoglobin saturation as measured by pulse oximetry (S_{pO_2}) decreased to 85% but returned to 95% when positive pressure ventilation was resumed. ABG values on 50% were pH 7.48, P_{aCO_2} 35 torr, P_{aO_2} 116 torr, and S_{aO_2} 99%.

On Day 12 the patient's respiratory status improved. The chest radiograph was consistent with increased lung volume and diminished interstitial markings. The patient was comfortable and in no distress. Airway pressure settings remained at 16 cm H_2O IPAP and 8 cm H_2O EPAP on F_{DO_2} of 0.40, with S_{aO_2} ranging from 93-97%. The absolute neutrophil count increased to 800/mm^3. On Day 13, the airway pressures were reduced to IPAP of 14 cm H_2O and EPAP 6 cm H_2O. F_{DO_2} was maintained at 0.40. ABG values at this setting were pH 7.49, P_{aCO_2} 23 torr, P_{aO_2} 109 torr, and S_{aO_2} 99%.

Noninvasive positive-pressure ventilation was discontinued on Day 14, and the child was placed on a 30% oxygen face mask. ABG values were pH 7.51, P_{aCO_2} 23 torr, P_{aO_2} 55 torr, and S_{aO_2} 92%. The chest radiograph suggested improved lung volumes, and the absolute neutrophil count was 2,000/

mm^3. The patient was discharged to the Hematology/Oncology Service with oxygen by face mask (F_{DO_2} = 0.30) with S_{pO_2} greater than 95%. He was subsequently discharged to his home.

Discussion

These cases illustrate the successful application of noninvasive, pressure-limited ventilation via a nasal mask in two children with acute respiratory failure. Several authors[2-7] have described the use of noninvasive positive-pressure ventilation (NIPPV) through a nasal mask in adults, but we have found no information regarding its applicability to children. The adult series describe patients with neuromuscular disease, acute exacerbation of chronic obstructive pulmonary disease, and hypoxemic respiratory failure of various etiologies. In these cases, nasal intermittent positive-pressure ventilation was used in conjunction with a system similar to the one that we used or with a conventional ventilator. Pennock et al[2] reported a 76% success avoiding intubation and conventional ventilation (22/29 patients) in the treatment of acute respiratory failure in patients who had not improved after diuresis, supplemental oxygen administration, and the use of bronchodilators. All patients were judged to require endotracheal intubation and mechanical ventilation at the beginning of the study. Waldhorn[3,4] reported the use of positive pressure (IPAP and EPAP) noninvasively in the successful management of 8 adult patients with hypoventilatory respiratory failure and nocturnal hypercarbia; four of these patients had obesity hypoventilation syndrome, whereas the remainder had neuromuscular disease.

Similar results were reported by Pennock et al,[5] Elliot et al,[6] and Wysocki et al.[7] Their patients consisted of adults with acute respiratory failure of various etiologies, including neuromuscular disease and atelectasis. Wysocki described NIPPV in conjunction with a Puritan-Bennett 7200 ventilator (Puritan-Bennett, Carlsbad CA) set in a pressure support mode. Ellis et al[8] described the use of intermittent positive pressure ventilation via a nasal mask in the treatment of hypoventilatory respiratory failure in a 6-year-old girl. Positive pressure was delivered intermittently at inspiration with an LP 3 ventilator (Lifecare, Lafayette CO). Bach et al[9] also used intermittent positive pressure via a nasal mask with a portable home ventilator in two adult patients with respiratory insufficiency resulting from neuromuscular disease.

Bersten et al[10] applied continuous positive airway pressure (CPAP) using a nasal mask in the management of 19 adult patients with acute cardiogenic pulmonary edema. This mode of ventilation produced rapid improvement in respiratory rate, acidemia, and oxygenation. Suter and Kobel[11] reported similar success in adults with respiratory failure but cautioned that respiratory failure as a result of sepsis, intrinsic lung disease, and heart failure may not be amenable to treatment with CPAP, the latter being of value only in postoperative or post-traumatic pulmonary dysfunction. Williamson and Modell[12] described the case of a 30-year-old with postoperative atelectasis who was successfully managed with CPAP applied intermittently with a face mask. However, the data by Bersten et al[10] and Meduri et al[13] demonstrate that CPAP can be used in patients with intrinsic lung disease. Four of the 10 patients in Meduri et al's report[13] had acute hypoxemic respiratory failure from a primary lung disease, whereas Bersten's data included 19 adults with cardiogenic pulmonary edema. The review by Branson et al describes this technique and its application.[14]

We believe, based on our experience with these two patients, that use of bi-level positive pressure ventilation via mask in children with acute respiratory failure may reduce the need for invasive mechanical ventilation. The response of our patients was similar to results in the adult studies cited despite differences in age. Although we alternated inspiratory and expiratory assistance (IPAP and EPAP), some clinicians[15] have applied inspiratory assistance either continuously or periodically using a face mask and conventional ventilator. Others[11] applied CPAP using an underwater seal or a 10-cm H_2O valve.[10] Unlike CPAP alone, positive pressure at different levels on inspiration and expiration provides a theoretical advantage in that the work of inspiration is assisted by providing pressure-support ventilation. CPAP systems can fail to provide sufficient flow or steady pressure, particularly with high levels of inspired oxygen[2] and also CPAP may result in more hemodynamic compromise than EPAP.[14]

Our first patient (Case 1) required IPAP in the range of 12-20 cm H_2O and an EPAP in the range of 6-8 cm H_2O to reverse the respiratory failure; the second patient (Case 2) required IPAP in the range of 10-16 cm H_2O and an EPAP in the range of 4-8 cm H_2O during treatment of respiratory failure. These settings gave our patients pressure support sufficient to ensure adequate gas exchange. The delivered tidal volume on positive pressure at two levels depends on the pressure boost, airway resistance, lung compliance, and inspiratory time, which, in the spontaneous mode is determined by the patient.[16] Our patients did not require more than a 6-12 cm H_2O pressure boost to deliver adequate tidal volumes.

The etiology of pulmonary edema and atelectasis in the first patient is unclear. Patients with Down's Syndrome are hypotonic and suffer from obstructive sleep apnea and nocturnal hypoxia, which if prolonged can result in cor pulmonale and pulmonary hypertension.[17] Patients in the recumbent position have a low FRC and increased closing volume, and are prone to atelectasis. Elliot et al[6] demonstrated that EPAP can increase FRC and reduce a tendency to atelectasis in patients with neuromuscular disease. Small atelectatic areas may be recruited with application of EPAP, minimizing ventilation-perfusion mismatch and improving oxygenation.

The need for prolonged endotracheal intubation and mechanical ventilation in neutropenic patients with hematologic malignancies has been associated with a poor outcome,[18-20] perhaps in part because of the increased vulnerability of the immunocompromised patient to infection associated with intubation and ventilation. Invasive mechanical ventilation was avoided in the patient with leukemia and neutropenia (Case 2) through the application of bi-level positive airway pressure with a nasal mask.

Adult patients receiving NIPPV have reported subjective improvement in dyspnea, coupled with improvement in measurable indices of respiratory function such as respiratory rate, tidal volume, ABG values, and S_{aO_2}.[10,21] Carrey[21] demonstrated that improvement of respiratory indices during nasal mask ventilation is caused by the reduction in inspiratory muscle energy expenditure. He reported a suppression of phasic electromyographic activity of the diaphragm and parasternal muscles, as well as the development of positive inspiratory pressure deflections with the initiation of NIPPV in patients with acute respiratory insufficiency. This suggests that nasal mask ventilation with IPAP and EPAP can reduce energy cost of breathing in respiratory failure in a way comparable to that observed in patients on conventional mechanical ventilation, by resting the respiratory muscles. Hill et al,[22] using positive pressure ventilation in the treatment of nocturnal hypoventilation and hypercarbia in adults with neuromuscular disease, postulated that improvement in gas exchange is the result of amelioration of nocturnal hypoventilation and the eventual reversal of the "central fatigue" that blunts central respiratory sensitivity to carbon dioxide retention. The amelioration of tachycardia and tachypnea and the reduction in the use of accessory muscles of respiration seen in our patients following institution of noninvasive positive-pressure ventilation is more likely the result of respiratory muscle rest than of resetting of the central respiratory mechanism because chronic hypercarbia was not a problem in these cases.

Intolerance of nasal mask ventilation was not a problem in our patients. The reasons for failing noninvasive positive-pressure ventilation in Pennock et al's report[5] included failure to tolerate the mask, confused and combative patients, failure to relieve hypoxemia, and the presence of excessive secretions. In addition, the patient should be evaluated for adequacy of cough and gag reflexes to minimize the potential for aspiration while on mask ventilation. Our first patient (Case 1) wore the mask continuously on each occasion for an average of 3.5 days. Excoriation of the skin on his face was observed, a problem similar to that reported by Pennock et al.[2] This problem can be ameliorated by applying a patch of wound care dressing to the nasal bridge before instituting the mask, as suggested by Pennock et al.[2] Periodic examination of the face for excoriation was done in the second patient (Case 2), based on our experience with the first. Bach[23] remarked that excoriation of the nasal skin should not be regarded as an impediment to the use of nasal mask ventilation because there are commercially available interfaces (Sefam Mask, Lifecare, Lafayette CO) that are effective and convenient for the entire spectrum of bi-level positive airway pressure, CPAP, and nasal intermittent positive pressure ventilation applications.

Endotracheal intubation with its attendant risk of increased morbidity was avoided in the second patient (Case 2) under clinical circumstances that otherwise would have made intubation inevitable. In the first patient (Case 1), reintubation after an initial 2 weeks of endotracheal intubation was avoided. Apart from these advantages, we cannot conclude that noninvasive bi-level positive pressure ventilation is superior to endotracheal intubation and conventional mechanical ventilation. Although its use is well established and described in adults, more controlled clinical studies are required in the pediatric population before widespread use can be recommended for pediatric patients in acute respiratory failure.

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RETROSPECTROSCOPE REDUX:

How To Make Hasenpfeffer

In July 1974, Congress amended the Public Health Service Act to establish National Research Service Awards. In this 1974 "National Research Act," Congress declared that "the success and continued viability of the Federal biomedical and behavioral research effort depends on the availability of excellent scientists and a network of institutions of excellence capable of producing superior research personnel." In short, Congress re-established NIH Research Fellowships and Research Training Programs. But with an important change—it asked the National Academy of Sciences to determine, year by year, the nation's over-all need for biomedical personnel, the subject areas in which research personnel are needed, and the number of personnel needed in each area. Congress also instructed the Secretary of HEW to make Fellowship and Training Awards to fill only these documented needs.

This seems to make sense. Until 1975, the number of Research Fellowships and Traineeships was determined by the law of supply and demand: *supply* for the most part meant how much money Congress appropriated and the National Institutes of Health allocated for training awards (and for research of newly trained scientists), and *demand* meant how many young men and women wanted a full-time or part-time career in medical research. Now the number trained will be determined by national need. For example, what needs to be decided for the lungs is what problems remain to be solved, the urgency of each, and how many scientists with what special training are needed to solve them. Then Congress will appropriate enough dollars to train the precise number of the right kinds of scientists, and the right kinds of young men and women will apply, be selected for research training, enter the training pipelines, come out in three to five years, and start turning out the right answers.

Now of course it isn't this simple. Every branch of medicine will want all of its problems solved all at once and there won't be enough dollars for trainee stipends or enough qualified candidates, enough trainers for the trainees, enough laboratories for them to work in, or enough research dollars in three or four years to pay for their research once they are established, independent scientists! And anyone who's been in the research business very long knows that not every trainee who looks good in the interview ought to do research at the end of his or her traineeship; indeed, one of the main purposes of putting people to work as research trainees is to see who's likely to succeed and who's not. So that means feeding more trainees into training programs that will be suited for research careers and likely to survive in the competition for research dollars. And the selection of the right number of trainees also depends on how many who are now in the research business retire, or die, or become deans.

I suppose all these problems that relate to numbers can be fed into computers and solved. But how do we get the right people to work on the right problems? Here the Retrospectroscope should tell us how to go about it. All we need to do is to identify 500 or 1,000 scientists who are already accepted as "superior research personnel," look into our Scope and see how they connected with the "right" field, i.e., that in which they made their important contribution. I've just completed step one of a pilot run. My plan couldn't be simpler: I said, "I'll collect the list of publications of some scientists who are highly regarded for their contributions to pulmonary or respiratory science and see how they entered scientific research. The easiest way to learn how they began is to read the first published scientific paper of each; I'll then know how to start the

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Table 1. Titles of the First Published Scientific Article of Forty-Three Scientists Whose Names Appear Alphabetically in Table 2.

1. Hereditary elliptocytosis associated with increased hemolysis.
2. The gaseous metabolism of the submaxillary gland.
3. Intrapulmonary mixing of helium in health and in emphysema.
4. The action of ouabain (G-strophanthin) on the circulation in man, and a comparison with digoxin.
5. Factors promoting venous return from the arm in man.
6. Blood coagulation in haemorrhagic diseases.
7. The effect of testosterone proprionate on the arginase content of the liver, kidney, and intestine.
8. Metabolism of leucocytes taken from peripheral blood of leukemic patients.
9. Les variations du sucre sanguin a la suite de l'injection intraveineuse de novarsénobenzol.
10. The respiratory rate and ventilation in the newborn baby.
11. The vaso-dilator action of potassium.
12. Syndrome de Looser, ostéopathie de famine et ostéomalacie.
13. Experimental studies on heteroplastic bone formation.
14. Fatty acid components in the unfertilized egg of arbacia punctulata.
15. Ueber die Bildung von Oxyden mehrwertiger Metalle aus ihren Hydroxyden.
16. Gas stores of the body and the unsteady state.
17. Salt antagonism in gelatine.
18. The medical use of thiocyanates in the treatment of arterial hypertension.
19. The measurement of intraesophageal pressure and its relationship to intrathoracic pressure.
20. The relation of philosophy to science.
21. On the morphology and mitosis of *Trichomonas buccalis*.
22. Pathogenesis of so-called diffuse vascular or collagen disease.
23. A propos d'un nouveau filtre sanguin à grand débit.
24. Influence de la teneur en calcium et en potassium du liquide céphalo-rachidien sur le système vasomoteur.
25. A note on the papillary adenoma of the corpus uteri.
26. The inhibition of frostbite wheals by the iontophoresis of anti-histaminic agents.
27. *Klebsiella* in respiratory disease.
28. Effect of certain drugs and ions on the oyster heart.
29. The surface tension of aqueous solutions of dipolar ions.
30. Demonstration of apparatus for recording muscle and nerve action potentials.
31. Histochemical studies on the lens following radiation injury.
32. Effect of cyanide on respiration of the protozoan, *Colpidium Campylum*.
33. Endobronchial tuberculosis.
34. Structure and function of placenta and corpus luteum in viviparous snakes.
35. Diet determinations. A graphic method.
36. Sulfapyridine therapy in pneumonia: Discussion of apparent failures and complications.
37. The reaction of vitamin A with Lieberman-Burchard reagent.
38. Calcium determination by flame photometry; method for serum, urine, and other fluids.
39. Demonstration of anatomy of giant fiber system of squid by microinjection.
40. Observations on the physiology of the Lepidopteran heart, with special reference to reversal of the beat.
41. Zur Kenntnis der Differenzierungsvorgänge im Epithel des Ductus cochlearis.
42. Measurements of the ventilation-perfusion ratio inequality in the lung by the analysis of a single expirate.
43. Ligation of inferior vena cava.

next generation on the right track." In Table 1, I've collected the titles of the first articles published by 43 scientists whose names are listed alphabetically in Table 2* (along with a few words identifying each with one or more fields of pulmonary or respiratory research).

Let's look at the titles in Table 1. Only seven of the 43 titles really deal with respiration or the lungs—nine if we consider respiration in the very

*This list is not intended to represent my Roll of Honor or Hall of Fame since this would include a hundred or more names. My list does, however, include a broad sample: it contains some internists, pediatricians, anesthetists, anatomists, pathologists, physiologists, pharmacologists, biophysicists, bioengineers, four deans, three Nobel laureates (and some others who also deserved to be), the NHLI Director for Lung Diseases, and a few presidents of the American Thoracic Society. Regardless of the category, each has done superior research.

Table 2. Alphabetical List of Scientists; the Title of the First Published Article of Each Appears Somewhere in Table 1.

(Phrases in parentheses refer to area of best-known pulmonary or respiratory research of each scientist.)

1. Mary Ellen AVERY (Respiratory distress syndrome; lung metabolism)
2. Joseph BARCROFT (Oxygen and hemoglobin)
3. David V. BATES (Clinical pulmonary physiology)
4. William A. BRISCOE (Distribution of pulmonary ventilation and perfusion)
5. E. J. Moran CAMPBELL (Dyspnea; mechanical factors in breathing)
6. Ronald V. CHRISTIE (Emphysema; elastic properties of lungs; pulmonary function)
7. Leland C. CLARK (P_{O_2} electrode; artificial heart-lung; artificial blood)
8. John A. CLEMENTS (Pulmonary surfactant; respiratory distress syndrome)
9. André COURNAND (Intrapulmonary gas mixing; ventilation/perfusion ratios; clinical pulmonary physiology)
10. Kenneth W. CROSS (Respiration in newborn babies)
11. Geoffrey S. DAWES (Fetal and neonatal respiration and circulation; chemoreflexes)
12. Pierre DEJOURS (Chemoreflexes; regulation of breathing; exercise)
13. William DOCK (Ventilation and blood flow to lung apices)
14. Arthur B. DuBOIS (Body plethysmograph; pulmonary blood flow clinical pulmonary physiology)
15. U.S. von EULER (Hypoxia and pulmonary arteriolar constriction regulation of breathing)
16. Leon E. FARHI (Pulmonary and tissue gas exchange)
17. Wallace O. FENN (Pressure-volume curve; $O_2 - CO_2$ diagram)
18. Robert E. FORSTER (Pulmonary diffusing capacity; rapid reactions of hemoglobin)
19. Donald L. FRY (Pressure-volume-air flow relationships)
20. John S. HALDANE (Carbon dioxide and regulation of breathing)
21. H. Corwin HINSHAW (Chemotherapy of tuberculosis)
22. Jerome I. KLEINERMAN (Experimental pathology of pulmonary disease; emphysema)
23. Claude J. M. LENFANT (Comparative respiratory physiology; regulation of $Hb - O_2$ affinity)
24. Isidoor R. LEUSEN (Cerebrospinal fluid and regulation of breathing)
25. Averill A. LIEBOW (Pulmonary and bronchial circulations; lung pathology)
26. Jere MEAD (Mechanics of breathing)
27. Jay A. NADEL (Mechanisms of airway constriction; tantalum bronchograms)
28. Arthur B. OTIS (Work of breathing; hypoxia; comparative physiology)
29. John R. PAPPENHEIMER (Cerebrospinal fluid and regulation of breathing)
30. Richard E. PATTLE (Alveolar lining layer and surfactant)
31. Solbert PERMUTT (Mechanics of ventilation and of pulmonary blood flow)
32. Robert F. PITTS (Organization of the respiratory centers)
33. Donald F. PROCTOR (Upper and lower airways; air flow; pleural pressure)
34. Hermann RAHN (Gas exchange; inert gases; environmental physiology)
35. Dickinson W. RICHARDS (Pulmonary function; uneven alveolar ventilation; clinical pulmonary physiology)
36. Richard L. RILEY (Diffusing capacity; ventilation-perfusion ratios; airborne infection)
37. Eugene D. ROBIN (Intracellular acid-base metabolism and gas exchange; regulation of breathing; pulmonary embolism)
38. John W. SEVERINGHAUS (P_{CO_2} electrode; regulation of respiration; high altitude physiology)
39. Norman C. STAUB (Pulmonary capillaries, lymph and edema; pulmonary structure-function)
40. S. Marsh TENNEY (Adaptations to high altitude; regulation of breathing)
41. Ewald R. WEIBEL (Morphometry of the lung)
42. John B. WEST (Uneven distribution of gas and blood; gas exchange)
43. James L. WHITTENBERGER (Artificial respiration; mechanics of breathing)

broadest sense. But even more astonishing is that in only five instances does the title of the first published article deal with the research area in which the scientist eventually made his important contributions! I estimate that a National Committee charged with selecting appropriate research topics for pulmonary or respiratory fellows going into the 1975 training pipeline (to meet 1978 national needs) would reject 35 of the 43 areas of research. And I believe that a 1975 Congress would be concerned if the NHLI awards a future pulmonary scientist a traineeship to study arbacia eggs, the oyster heart, viviparous snakes, the squid, or the lepidopteran heart. But since we're looking at the genesis

of successful pulmonary scientists, there must be a lesson here. The lesson seems to be that the first step in recruiting a talented, creative scientist is to get him involved in science, in any science, in any field. (As Professor Carl Schmidt used to say "In making hasenpfeffer, the first step is to catch the rabbit!") In their first paper, only seven scientists dealt with lungs or pulmonary ventilation. Four more entered the field with their second paper, an additional ten by their fifth paper, another six by their tenth paper, five more by their fifteenth, and another four by their twentieth. The other seven came aboard after twenty or more publications in other fields. Fenn had already distinguished himself in two other fields (muscle contraction and potassium metabolism) before he became a pulmonary physiologist.

To make manpower planning in specific areas even more complex, only about half of those on my list limited their research to the lungs and respiration once they entered the field. The other half occasionally, or often, worked in other areas and some left the field completely. Fry now works largely on experimental atherosclerosis. Pitts, after his classic studies on the respiratory centers, gained greater fame as a renal physiologist. U.S. von Euler, after his very important work on regulation of breathing and the effects of hypoxia on the pulmonary circulation, won the Nobel Prize for his studies on norepinephrine and then went on to work on prostaglandins. Dickinson Richards was one of the giants in pulmonary physiology well before he (with Cournand) won the Nobel Prize for cardiovascular studies using the cardiac catheter. Einthoven (not on my list) did important work on the mechanics of breathing in 1892 before he invented the electrocardiogram.

My pilot study is open to many criticisms: It has looked at only those in the forefront of their field and not at those equally needed in science to confirm and extend the new discoveries and tie up the loose ends; it has looked only at those who entered scientific careers in an earlier era; it has *not* looked at those who tried their hand at research and decided that it was not for them. But even more important, this study gives no inkling of why each of the 43 gave science a try in the first place, who and what made it possible for him or her to stay in science, and why each initially or much later decided to work on pulmonary and respiratory problems. I know the answers to these why's, who's, and what's for my own career (see Addendum # 2) but not for the 43 scientists in Table 2—even for those whom I've known well for many years. Factors influencing career decisions and directions are usually quite complex.

What can we learn by this look into the Retrospectroscope? First, that it is essential to get creative young men and women actually involved in research. Any research? Probably yes, because it seems that the first research project is for most young scientists a time for deciding whether research is the right career choice. Second, the young scientist should be encouraged to learn broadly in science, in anticipation of a change, sooner or later, in his research interests. And third, a field looking for research recruits will have no trouble attracting them if there are exciting and challenging problems to be solved.

JULIUS H. COMROE, JR.

Addenda

1. Sorry—I forgot to tell you which scientist in Table 2 wrote which article whose title is in Table 1. The order is the same in both tables: Avery wrote # 1, Barcroft # 2, and so on down to Whittenberger and # 43.
2. The title of *my* first paper had nothing to do with respiration or the lungs. It was "Further studies on the pharmacology of acetyl β-methyl choline and the ethyl ether of β-methyl choline." I did the work in the summer vacation between my second and third years of medical school, instead of spending the summer as usual playing tennis in my hometown of York, Pennsylvania. Why? Because I was engaged to marry a Philadelphia girl and I wanted to stay in Philadelphia. Simple enough. The answer to "Why did I stay in science?" is more complex, as indeed I suspect it is with most of us.

Arrest following a Prolonged Course of Periodic Coughing and Fever in a Child

Lynn M (Rasmussen) Sullivan RRT and Robert M Kacmarek PhD RRT

A 1-year-old boy was referred to our institution from an outlying hospital. The patient had experienced intermittent low-grade fever with daily coughing episodes productive of thick yellow sputum for 2-4 months prior to referral. Three days before admission, the child had spiked a temperature of 101°F, and a chest radiograph revealed a left-lower-lobe pneumonia. On the morning of referral, during a coughing episode, the child became limp and cyanotic. Upon arrival in the emergency ward of the outlying hospital, the child demonstrated agonal breathing and was intubated. Marked difficulty was noted during manual ventilation, and high peak inspiratory pressures (> 50 cm H_2O) were required to adequately ventilate him. Approximately 10 min after intubation, the child suffered a cardiac arrest, was treated with appropriate drug therapy, and was transferred to our institution.

When he arrived at our institution, the child was being ventilated at rate 50/min, V_T 150 mL, F_{IO_2} 0.70, without PEEP. The child was on intracranial pressure precautions (attempted hyperventilation and head of bed raised 45°), his pupils were fixed and dilated, his blood pressure was being supported with inotropic agents (dopamine, dobutamine), and blood gas values were P_{aO_2} 70 torr [9.31 kPa], P_{aCO_2} 52 torr [6.93 kPa], and pH 7.29. Breath sounds were markedly decreased on the right, and crackles were heard over the entire left lung field. A chest radiograph (Fig. 1) was obtained.

Ms Sullivan is a Staff Therapist and ECMO Specialist, Respiratory Care Services, Massachusetts General Hospital; Dr Kacmarek is Assistant Professor, Department of Anesthesiology, Harvard Medical School, and Director, Respiratory Care Services, Massachusetts General Hospital—Boston, Massachusetts.

Fig. 1. Chest radiograph from 1-year-old boy taken on arrival from emergency ward of outlying hospital. Patient had suffered cardiac arrest following a coughing episode.

Questions

Radiographic Findings: How would you interpret this chest radiograph?

Possible Pathophysiology: What pathophysiologic process could account for this presentation?

Further Procedures: What further diagnostic or therapeutic procedures are indicated?

Answers and Discussion on Next Page

Answers

Radiographic Findings: The endotracheal tube is properly placed with the tip 3.5 cm above the carina. There is marked hyperinflation of the right lung and a shift of the mediastinum to the left as indicated by the heart shadow located left of the vertebral bodies. There is atelectasis, collapse, or consolidation in the left lower lobe, as indicated by loss of the diaphragm shadow on that side; this could be indicative of an infiltrative process such as pneumonia. The lingula also appears to have a small infiltrate.

Possible Pathophysiology: Considering the lengthy presentation and productive cough, the marked and rapid deterioration, and the inability to ventilate, foreign-body aspiration should be suspected.

Further Procedures: A bronchoscopy for diagnosis and possible therapy should be performed.

Discussion

Flexible fiberoptic bronchoscopy performed at the bedside identified a foreign body in the right main-stem bronchus. The child was taken to the operating room where a rigid bronchoscopy was performed. A small piece of wood, believed to be a fruit stem, was removed from the right main-stem bronchus where a large amount of exudate was observed. The left main-stem bronchus was inflamed, with granulomatous tissue present. It appeared that the child had aspirated the foreign body into the left main-stem bronchus and that the foreign body had remained there for a lengthy period of time, resulting in obstructive pneumonia. On the day of admission, it is believed that the child coughed the foreign body out of the left main-stem bronchus but it subsequently lodged in the right main-stem bronchus, limiting ventilation to the right lung and necessitating high peak airway pressure during ventilation. Although the foreign body was successfully removed, with a marked reduction in peak airway pressure, brain damage suffered during the cardiac arrest was irreversible, and the child subsequently died.

Treatment of foreign-body aspiration in the small child involves establishing a patent airway and removing the foreign body with rigid bronchoscopy under general anesthesia in the operating room.[1] Solid body removal is generally uncomplicated if the airway is stable; however, semisoft objects that tend to break (eg, peanuts, beans) are more difficult to remove because pieces tend to break off and travel peripherally during attempted removal.

Symptoms of foreign-body aspiration are variable and are affected by location and type of foreign body, degree of obstruction, and length of time between aspiration and presentation. Organic foreign bodies are more likely to cause an inflammatory reaction than inorganic ones.[1] Partial obstruction caused by a foreign body can become a total obstruction when tissue edema develops. This can shorten the asymptomatic period experienced by the patient and lead to an earlier abnormal physical examination when compared to the patient with an inorganic foreign-body aspiration.[1]

Location and degree of obstruction may also vary. With complete obstruction, no air movement occurs distal to the foreign body. If complete tracheal obstruction occurs, no breath sounds can be heard; whereas if bronchial obstruction occurs, breath sounds are absent on the affected side. A so-called ball-valve type of obstruction generally allows inspiration; however, no breath sounds are heard on expiration and hyperinflation may become apparent. Partial airway obstruction generally presents with wheezing, cough, dyspnea, or decreased breath sounds over the affected area.

Of the 3,000 deaths per year in the U.S attributed to foreign-body aspiration, 20% are in children less than 4 years old.[2] The diagnosis of foreign-body aspiration is made within 24 hours of the event in about half of the cases, and within 3 days in greater than 93% of cases.[2,3] The complication rate dramatically increases if diagnosis is delayed beyond 3 days.[3] In about 6.7% of the cases, the diagnosis is made more than 30 days after the event.[3] Delay in diagnosis is frequently a result of presentation resembling more benign problems[4] and may lead to chronic complications such as recurrent pneumonia, abscess, or bronchiectasis.[1]

Treatment with antibiotic and steroid medications can inhibit reaction to the foreign body, leading to a false sense of security and reinforcing the faulty diagnosis.[2,3] Although 50% of children with foreign-body aspiration have a history of choking and periodic coughing, parents may not suspect foreign-body aspiration because children are usually free of symptoms between coughing episodes.[3,5] The mortality rate for foreign-body aspiration is 0-2%[5] but increases as time between the event and diagnosis increases and serious complications develop.[1,3-5] Death has been reported when a foreign body in a main-stem bronchus dislodges and proceeds to obstruct the opposite bronchus, as in this case.[6] In any child presenting with cough and wheezing, foreign-body aspiration should be suspected.[2,5]

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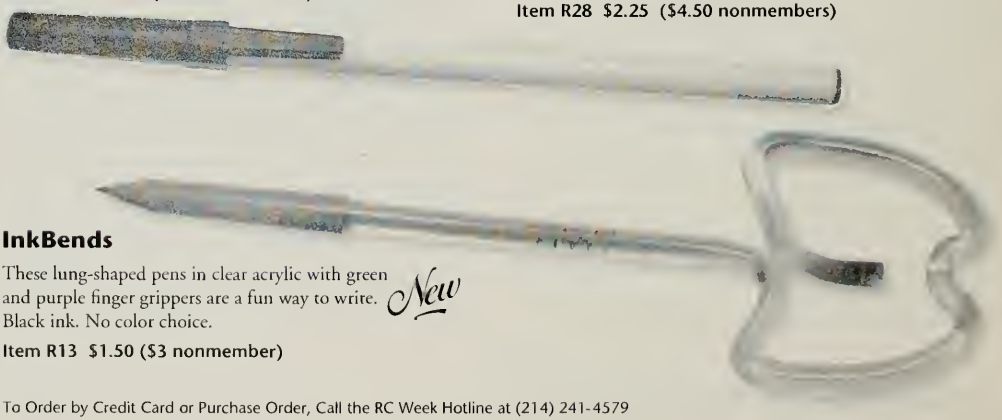

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Blood Gas Corner #31— A Patient Presenting with Leg Pain and Shortness of Breath

Joseph E Ollivier RRT

A 59-year-old man presented with acute dyspnea, right-side pleuritic chest pain, and blood-tinged sputum. He stated that his dyspnea had progressively worsened over the last 3 or 4 days and he had suffered right-leg pain over the 24 hours prior to admission. His vital signs were blood pressure 134/76, pulse 128/min, and respiratory rate 36 breaths/min. He was alert, slightly diaphoretic with strong peripheral pulses, and afebrile. The rest of the physical exam was unremarkable. A chest radiograph revealed normal lung fields with no cardiomegaly. The results of arterial blood gas analysis, drawn while the patient breathed room air, were pH 7.37, P_{aCO_2} 10 torr [1.33 kPa], P_{aO_2} 49 torr [6.53 kPa], HCO_3^- 6 mEq/L [6 mmol/L], base excess −15 mEq/L [−15 mmol/L], and S_{aO_2} 89%. Routine spirometry and EKG were essentially normal. Other laboratory tests, including serum lactic acid, and results of a complete blood count, were also within normal ranges.

Mr Ollivier is Chief, Respiratory Therapy Department, Veterans Administration Medical Center, Fresno, California.

Study Questions

1. How would you interpret the initial results of the blood gas analysis?

2. What diagnosis might explain this patient's blood gas data and clinical presentation?

3. What other information or diagnostic tools would aid in the diagnosis and management of this patient?

Answers and Discussion on Next Page

Answers

1. Interpretation. The blood gas interpretation has several possibilities, including a fully compensated respiratory alkalen ia, a compensated metabolic acidemia, or a combined alkalemia-acidemia; the oxygen status is consistent with a moderate-to-severe hypoxemia with a widened alveolar-arterial tension gradient. There is an increase in alveolar ventilation. When the peripheral chemoreceptors in the carotid bodies sense a decrease in the partial pressure of oxygen in arterial blood, they are stimulated to increase alveolar ventilation in an attempt to increase the P_{aO_2}.[1] The resulting high alveolar ventilation lowers the P_{aCO_2}, resulting, initially, in an elevated pH. To compensate, the unaffected system restores pH to normal by increasing renal excretion of bicarbonate, retaining chloride, and reducing both the formation of ammonium ion (NH_4^-) and the excretion of the second ionization of phosphoric acid (HPO_4^{2-}). The resultant blood bicarbonate level brings the acid-base ratio back toward 20:1, thus normalizing pH. When respiratory alkalosis is compensated, $[HCO_3^-]$ will have decreased 5 mEq/L [5 mmol/L] for every 10 torr [1.33 kPa] decrease in P_{aCO_2}.[2]

In metabolic acidosis (such as lactic acidosis or ketoacidosis), the arterial pH depends on the rate at which acid is being produced and on alveolar ventilation. As the acidosis develops, ventilation increases and P_{aCO_2} falls. The fall in P_{aCO_2} creates a reduction in normal chemical stimuli and inhibits the degree of ventilatory compensation because when CO_2 tension is high the carotid bodies are more sensitive to the partial pressure of oxygen. This sensitivity is reversed when P_{aCO_2} is low. A metabolic acidosis can be of two types, one that is associated with an increased anion gap and one with a normal anion gap (8-16 mEq/L [8-16 mmol/L]). Anion gap has been defined as the abnormal excess of negatively charged ions (anions) in solution—blood.[3] However, an excess of anions in solution never really exists; electrical neutrality is maintained. The term anion gap is one of convenience and is estimated by subtracting the sum of the concentrations of the major anions (HCO_3^- and Cl^-) from the concentration of the major cation (Na^+), that is

$$[Na^+] - ([HCO_3^-] + [Cl^-]).$$

Anion gap is usually caused by the presence of anions, other than bicarbonate, in large quantities. A metabolic acidosis with an increased anion gap is associated with uremia, diabetes, paraldehyde poisoning, lactic acidosis, and methanol, ethanol, and salicylate intoxication. Metabolic acidosis with a normal anion gap is associated with diarrhea and acetalzolamide-induced and renal-tubular acidoses.[2] This patient had none of the conditions associated with a normal anion gap.

2. Diagnostic Possibilities. Many diseases may cause alveolar hyperventilation. In all cases, the fall in P_{aCO_2} is caused by three basic physiologic conditions that occur separately or in combination with one another: (1) an arterial oxygen deficit severe enough to stimulate the peripheral chemoreceptors; (2) a metabolic acidosis (such as lactic acidosis or ketoacidosis); and (3) abnormal stimulation of the ventilatory centers of the central nervous system. In this instance the patient's clinical presentation, afebrile condition, normal-range serum lactic acid, anion gap, and chemistry values suggest that the profound hypoxemia was likely due to pulmonary embolism.[2]

The most common symptoms of pulmonary embolism are chest pain and dyspnea; hemoptysis and diaphoresis occur in up to one third of the cases.[3] Tachypnea and tachycardia are observed in the majority of patients with pulmonary embolism but may be transient. Other features infrequently noted in patients with pulmonary embolism are fever, rales, wheezing over lung fields, cardiac murmur, gallop rhythm, phlebitis, abdominal pain, syncope, and cyanosis.[4,5] The most important information required to make the diagnosis of pulmonary embolism is the presence of predisposing risk factors for thrombus formation (eg, history of thrombophlebitis, obesity, prolonged immobilization, hyperviscosity syndromes), particularly because these patients may be symptom-free.[6]

3. Other Information. Currently the most commonly used procedure to diagnose pulmonary embolism is the ventilation-perfusion ($\dot{V}/\dot{Q}$) scan. When clinical findings are suggestive and the lung scan shows a high probability of pulmonary embolism, the diagnosis is accurate in about 90% of cases.[7] When coexisting lung disease prevents accurate interpretation of $\dot{V}/\dot{Q}$ scans, the pulmonary angiogram is the accepted reference standard for the diagnosis of pulmonary embolism. In this procedure, a catheter is inserted into the femoral vein and positioned to inject contrast material into the pulmonary artery. This technique is used to identify an intraluminal filling defect or an interruption in blood flow.[8]

Discussion

Pulmonary embolism is mechanical obstruction of a branch or branches of the pulmonary vascular tree. As ventilation continues, the result is a ventilation-to-perfusion mismatch that may be detected on a $\dot{V}/\dot{Q}$ scan. Hypoxemia is the clinical manifestation of the inadequacy of the other areas of the lung to which blood has been shunted. The majority of emboli come from the deep veins in the pelvis and legs.[9] Condi-

tions that predispose to thrombus formation are (1) vessel damage due to phlebitis, (2) hypercoagulation states, and (3) blood flow abnormalities. Primary hypercoagulation states are difficult to diagnose. Secondary abnormalities of coagulation and fibrinolysis include pregnancy, malignancy, nephrotic syndrome, liver disease, and the use of oral contraceptives.[10] Blood-flow abnormalities that contribute to thrombus formation include stasis and hyperviscosity. Blood-flow stasis may be due to obesity or prolonged immobilization. Hyperviscosity abnormalities include polycythemia, leukemia, and sickle cell disease.[11]

Heparin anticoagulation is considered primary therapy for most patients with pulmonary embolism. Initially it is administered as a bolus of 100 U/kg (total dose 5,000 to 10,000 U) followed by a continuous infusion of 800 to 1,000 U/hour. Activated partial prothrombin time should be maintained at 1.5 to 2.5 times published normal values.[11] Anticoagulants are administered orally to prevent recurrent thrombus formation. Warfarin, overlapping the heparin therapy by 4 or 5 days, is given orally for at least 3 months or indefinitely if risk factors such as cancer or massive obesity have not been resolved. The target range for the corrected prothrombin time with warfarin therapy is an INR (International Normalized Ratio) of 2.0 to 3.0.[10] Definitive treatment may include (1) thrombolytic therapy with streptokinase, urokinase, or tissue plasminogen activator to promote dissolution of thrombi in those patients with hypotension secondary to massive pulmonary emboli or (2) pulmonary embolectomy for patients who remain in shock despite thrombolytic therapy and supportive care. Patients who cannot tolerate orally administered anticoagulant therapy may require a surgically placed inferior-vena-cava filter to intercept emboli on their way to the lung.[12]

In this patient, an angiogram, performed shortly after admission, confirmed the presence of pulmonary embolism. The patient was treated with a heparin bolus of 10,000 U followed by a continuous infusion of 1,000 U/hour. Warfarin therapy, 10 mg daily, was started 4 days before discontinuing heparin. This dose was reduced to 5 mg/day at discharge and continued for 6 months. The patient was monitored closely during this period. He has since been asymptomatic and in good health.

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Guidelines for Pulmonary Rehabilitation Programs, by the American Association of Cardiovascular and Pulmonary Rehabilitation (AACVPR), edited by Gerilynn Connors BS RCP RRT and Lana Hilling RCP, authored by Committee Members C William Bell PhD MBA, Howard M Kravetz MD, Kathleen Morris RN MS RRT, and Andrew L Ries MD. Softcover, illustrated, 141 pages. Champaign IL: Human Kinetics, 1993. $28.00.

Pulmonary rehabilitation is a treatment modality involving a broad range of healthcare professionals. It has been defined as an 'art' because of the need to individualize program goals and treatment regimens. Pulmonary rehabilitation programs vary considerably in their content, structure, and duration. For practical reasons, many programs emphasize exercise training only. Unfortunately, this lack of uniformity has compromised development of the field of pulmonary rehabilitation, making it difficult to compare results between programs. **Guidelines for Pulmonary Rehabilitation Programs** is a 'how-to' reference for healthcare professionals interested in developing or enhancing their pulmonary rehabilitation programs.

The first section of the book outlines the components of a rehabilitation program, with seven chapters covering selection and initial assessment of rehabilitation candidates, patient training, exercise testing and training, psychosocial components, patient outcomes, and program management. The chapters are written in outline form with limited text but are amply supplemented with tables that emphasize the important points; unfortunately, the book includes numerous illustrations without legends, which contribute little to the reader's understanding of the subject. The appendix, the second half of the book, includes the full reproduction of the position paper of the American Association of Cardiopulmonary Rehabilitation written by Andrew Ries MD and the American Thoracic Society position paper. Two case studies by the editors complete the book. The chapters are well referenced; furthermore, the book includes a detailed list of educational materials and information on how they may be obtained.

Reimbursement remains one of the major impediments to the development of pulmonary rehabilitation. Unfortunately, the book offers little direction on how to deal with reimbursement issues. The editors also rely heavily on a small number of publications. Specifically, numerous tables are reproduced from the book entitled, **Pulmonary Rehabilitation: Guidelines to Success,** edited by JE Hodgkin, EG Zorn, and GL Connors. We did not review this book simultaneously and we cannot comment on the relative advantages of the two texts.

Although the authors did fail to address the reimbursement issues and to provide 'more bang for the buck' by including informative figure legends, **Guidelines for Pulmonary Rehabilitation Programs** represents a solid contribution to a field that needs increased standardization of caregiving modalities.

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Respiratory Care: A Guide to Clinical Practice, 3rd edition, edited by George E Burton MD, John E Hodgkin MD, and Jeffrey J Ward MEd RRT. Hardcover, 1,043 pages, illustrated. Philadelphia: JB Lippincott Co, 1991. $150.00.

This classic textbook for respiratory therapists is now in its third edition. Two major changes are obvious. The first is a reduction in the number of pages and the other a change in the authorship. The new edition has several new names, this time including anesthesiologists. A well-balanced mix of pulmonologists, respiratory therapists, and anesthesiologists now contribute to this text. The book retains its original layout and is divided into 3 sections and an appendix.

The first section is an introduction to the field of respiratory therapy, and it takes the reader through the history of the specialty, the role of the therapist in modern medicine, and the various aspects of training in and the delivery of respiratory care in the U.S.

Section 2 is the meat of the book—16 chapters on the science of respiratory therapy. It begins with pulmonary physiology and goes on to cover physical examination of the lungs. The extensive chapter on the chest x-ray is too detailed; a review of the general principles of x-ray diagnosis followed by a few common clinical conditions would serve just as well. Nevertheless, it contains a lot of essential clinical information on x-ray diagnosis. Airway management is handled well, again in great detail. If one might be allowed to be pedantic, Mallampati was misspelled (Mollampati). I find the discussion on assessment of the airway

rather vague. Mallampati's classification system of four oropharyngeal views of difficult intubations is not clearly presented, but this does not detract from an excellent discussion on airway management options. Mechanical ventilation, originally spread over four chapters, is now covered in two. Two chapters have been dropped entirely, the one on weaning (now part of the main chapter on ventilation) and the one on IPPB, which barely gets a mention—a reflection no doubt on its role in present-day respiratory therapy.

The third part of the book deals with the management of common respiratory conditions in the intensive care unit (ICU). These chapters all stand out for their clarity and organization. The principles of diagnosis and management are discussed first, followed by a brief discussion of the literature. The chapter on ARDS is good, although it does not mention permissive hypoventilation. The references are not as recent as one might have expected.

The final section of the book is a series of short monographs on a mix of topics. There is a good review of the physics and science of blood gas measurements, gas laws, and some rules-of-thumb for the management of the critically ill patient.

A variety of excellent books have been written on this subject; however, this book has much to offer and remains the standard in respiratory care education. It should find a place in any departmental library, but it is probably too expensive to be part of a personal collection. In addition to respiratory therapists, ICU residents should find it an excellent guide to respiratory intensive care.

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Deja Vu! A CPR Device to the Attention of MEDWATCH

Not only that, it's happening all over again![1] I was perusing a sheaf of items offered for sale by a company called Handsome Rewards (19465 Brennan Avenue, Perris CA 92599) when I came across an ad for a resuscitation aid called CPReviver. Because it was on sale for only $15.00 (discounted from $24.00), I was compelled to own one (Fig. 1).

The ad copy states "Prepare for emergencies of the most serious sort with the CPReviver. It's a necessity in today's world: a disposable contamination-free resuscitator that allows maximum air flow to and from the victim without dangerous backflow of possibly contagious fluids. Eliminates mouth-to-mouth contact while permitting lifesaving treatment for shock, cardiac arrest, drowning, smoke inhalation, drug overdose, convulsions, and other respiratory traumas." The directions (in both English and Spanish) instruct the potential rescuer to (1) have someone call for assistance; (2) part lips and clear mouth and throat; (3) place victim face up and tilt head back; (4) insert CPReviver over tongue and cover lips with mouthpiece; (5) pinch nostrils closed and blow deep breath into tube; (6) pause 4 seconds to allow victim to exhale through tube exhaust hole; and (7) repeat steps 5 and 6 every 4 seconds until breathing resumes or medical assistance arrives.

In examining this device, I wanted to determine how the one-way valve worked. It is a collapsible tube that opens only on the strength of the rescuer's breathing and then collapses on exhalation, diverting the expired gases out a side port. A quick test breath seemed to require substantial effort to open the valve. I placed a manometer in-line proximal to the valve and determined that it took about 30 cm H_2O pressure to open the valve; at higher flows, as much as 50 cm H_2O was required! Two additional facts emerged from my brief evaluation. First, the 30 cm H_2O was really a critical opening pressure—the valve remained closed to the patient below that pressure. Second, the CPReviver has a swivel feature that enables the rescuer to rotate the mouthpiece (rescuer) relative to the airway tube (victim). There was a noticeable air leak around this connection at various flows.

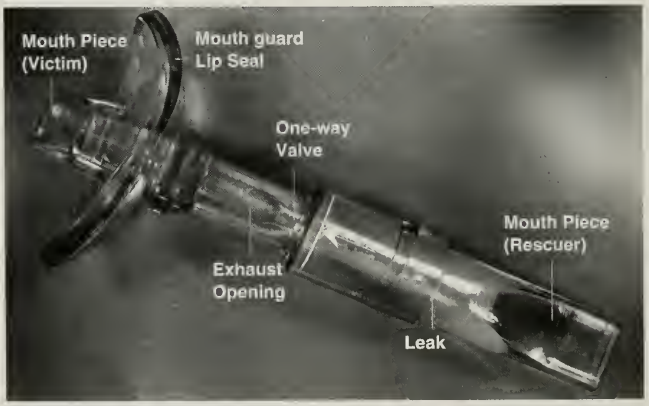

Fig. 1. CPReviver, showing one-way valve, exhalation port, rescuer and victim airway, and rescuer-victim-orientation swivel.

I believe that the marketing of this device at this time is irresponsible. In the hands of an untrained rescuer, it could endanger someone's life rather than save it. I haven't used this device on a person, but the results of my limited testing plus the less-than-adequate and brief directions contained on the packaging, lead me to believe that CPReviver is potentially dangerous. First, it is recommended for use on anyone age 4 or older. The directions say "blow deep breath into tube"—this action alone carries immense risk for a small child. Additionally, if someone does not generate the requisite 30 cm H_2O critical opening pressure for the valve, all the tidal volume would be delivered to the room, not the victim. The presence of a continuous leak of unknown size around the swivel connection further reduces the rescuer's ability to generate the opening pressure and deliver a reasonable tidal volume to the victim. Additionally, generation of the opening pressure requires rapid delivery of air (high flows), in direct opposition to current American Heart Association Guidelines for rescue breathing.[2]

The directions fail to emphasize the importance of maintaining the airway, aside from the initial instructions to tilt the head back. There also is no mention of monitoring the efficacy of one's efforts, as by chest rise or improvement in the patient's

color. Finally, the directions fail to address several 'What-if' questions such as: What if the victim vomits? What if the air is not going into the victim? What if the victim is already breathing?

I attempted to contact the manufacturer (Winspan Inc, Sylmar CA 91342) but I was unsuccessful (this company does not have a telephone listed). I notified the Medical Device and Laboratory Product Problem Reporting Program (now MEDWATCH).[3] In spite of the fact that the CPReviver is, as it states on the box, "Manufactured in Compliance with Government Regulations," One is inclined to ask, "What regulations?" I am convinced this is a device that in the hands of untrained individuals could result in otherwise avoidable injuries and deaths. Continued vigilance is necessary to find and expose devices such as CPReviver. We must watch with a critical eye, the endless parade of gimmicks and gadgets that attempts to lure us; some of these may lead to error and harm.

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Objections to Humidification Editorial

In their discussion of nosocomial pneumonia, Branson and Chatburn[1] dismiss the fine work of Gallagher et al[2] because it "disagrees" with three other studies. That results were not identical in studies employing different protocols, evaluating different patients, and conducted in different institutions is hardly surprising; but such differences are insufficient reason to dismiss research not conforming to the authors' apparent bias. In doing so, they confuse a Type I statistical error with a Type II error.

Two of the articles cited by Branson and Chatburn do support a decreased incidence of nosocomial pneumonia when the Pall heat-and-moisture exchanging filter (HME) is compared with hygroscopic condenser humidifiers (HCH).[3,4] That differences did not reach statistical significance in these studies simply reflects their relatively small sample sizes, but does not prove that no difference exists. Infections were not even evaluated in the third paper Branson and Chatburn cite in this section.[5] In that work Misset et al specifically state, "No attempt was made to assess the incidence of nosocomial pneumonia."

Branson and Chatburn make a number of errors when summarizing our study comparing an HME and various HCHs.[6] They specify that we used a tidal volume of 1,000 mL and a respiratory frequency of 10 breaths/min. Our paper indicates that the tidal volume was set to 10 mL/kg and the respiratory rate adjusted to provide an end-tidal P_{CO_2} near 35 torr. Neither tidal volume nor respiratory rate was specified in our paper, but typical values (during anesthesia) were 700 mL and 8 breaths/min.

The next sentence in Branson and Chatburn's editorial requires discussion. It states "The use of an HCH resulted in the delivery of an average of 29 mg H_2O/L, whereas the use of an HME yielded 19 mg H_2O/L." Although water loss per liter can be calculated using various assumptions, it is not what we reported. Our results are expressed in terms of percent water saved. A more serious concern is the carefully deceptive wording of this sentence, which implies that the average value for the four HCHs that we tested was 29 mg H_2O/L. However, that value actually refers to the *best* HCH; the *average* value was considerably less, and one of the HCHs saved *less* water than the HME. Our paper specifically concluded that differences among the tested units were not clinically important.

Branson and Chatburn's section discussing the influence of inspired gas temperature on body temperature contains so many errors that I find it virtually impossible to interpret. Even their conversion factors are wrong: 1 kcal/h equals 4,190 J/h (not 4.19 J/h as stated in the editorial), and 1 kcal/h equals 1.2 W (not 1.6 W as they state).

Minute ventilation and inspiratory and expiratory gas temperature and relative humidity are the only information required to calculate respiratory heat balance. These simple thermodynamic calculations have been reported numerous times. Branson and Chatburn's unique calculations require a series of assumptions, at least some of which are incorrect. They calculate respiratory heat loss from the fraction of total heat loss that is evaporative. As a result of confusing total evaporative loss (which includes insensible transcutaneous loss) with respiratory heat loss, they conclude that respiratory loss constitutes 21% of the total—roughly twice the correct value.[6,7]

The entire discussion of rewarming from accidental hyperthermia is irrelevant to typical clinical conditions. More importantly, it makes the implicit assumption that

changes in core temperature reasonably indicate changes in body heat content. It is well established that redistribution of heat within the body can markedly change core temperature without altering net heat balance.[8,9] Such changes are particularly well established during the initial rewarming period following accidental hypothermia.[10]

In summary, numerous errors in Branson and Chatburn's editorial make many of its conclusions suspect. They appear to have sacrificed considerable scientific objectivity in an effort to support their apparent bias.

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Dr Sessler has no financial interest in any anesthesia-related companies, nor does he consult for or accept honoraria from clinically related companies. His laboratory (UCSF Thermoregulation Research Laboratory) is supported by the National Institutes of Health and a number of companies including Augustine Medical Inc, Aspect Medical Inc, ICI Inc, Mallinckrodt Inc, Pall Corp, Gibeck Respiration Inc, Exergen Corp, and RSP Inc. During the last 5 years, the Laboratory has received $20,000 from Gibec Respiration Inc and $2,000 from Pall Corp.

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Mr Branson and Mr Chatburn respond:

Dr Sessler takes issue with several of the points in our editorial.[1] We will address each of his comments.

Dr Sessler suggests that it is our "bias" that the use of an HME/HCH does not reduce the incidence of nosocomial pneumonia. He cites the "fine" work of Gallagher[2] as evidence, but fails to discuss the limitations of Gallagher's study. As we have previously stated, this study utilized a retrospective control group and provides no group comparisons. The findings in this study are therefore difficult to confirm and are in conflict with other studies.[3-5] We are aware of the types of statistical errors seen in clinical studies when inappropriate numbers of patients are selected, and we understand that studies from different sites using different protocols often report different findings. The two articles we cite[3,4] do not compare HMEs to HCHs as Dr Sessler suggests but rather HMEs to heated humidifiers. In any event, these studies do not show a difference in the incidence of nosocomial pneumonia. Dr Sessler says they "do support a decreased incidence of nosocomial pneumonia," but that "differences did not reach statistical significance." You can't have it both ways. According to statistical convention, not reaching statistical significance means that the data do not support the conclusion that a difference exists. In contrast to the Gallagher et al study, these studies do provide group comparisons and allow scientific scrutiny of the methods. Roustan et al[3] state in their results "Incidence of nosocomial pneumonia *did not* significantly differ between Groups I and II (9/61 vs 5/55, $\chi^2 = 0.876$, $p > 0.25$). The study by Martin et al states "Seven percent of patients in the PUBCF and 19 percent in the HHWS developed a nosocomial bronchopneumonia during the study. This *did not* reach the level of significance."[4] As Dr Sessler says, Misset et al did not specifically address the incidence of nosocomial pneumonia.[6] As such, Misset et al's work supports neither his nor our viewpoints.

Dr Sessler does not explain how an HME reduces the incidence of nosocomial pneumonia and does not state whether he disagrees or agrees with our conclusion. Certainly the

literature demonstrates that nosocomial pneumonia is most frequently due to aspiration of oropharyngeal and/or gastric secretions,[7] and at least one group of authors state that nosocomial pneumonia caused by contaminated respiratory therapy equipment is infrequent.[8]

Perhaps we have missed Dr Sessler's point. He suggests we misrepresent the data to support our bias, yet he does not say he believes that nosocomial pneumonia is reduced or prevented by use of an HME. In fact, Dr Sessler seems to demonstrate his own bias by attributing the results of studies to Type I or Type II errors, depending upon which suits his fancy.

Dr Sessler is correct in that we mistakenly reported the test results in his and Dr Bickler's study.[9] The ventilatory setting should read a tidal volume of 10 mL/kg and respiratory frequency set to maintain an end-tidal CO_2 of 35 torr, not a tidal volume of 1,000 mL and frequency of 10 breaths/min. We apologize to the readers for our error. However, Dr Sessler's contention that we used "deceptive wording" raises our ire. Let us restate our interpretation of Bickler and Sessler's results in a manner that Dr Sessler should find straightforward and easy to comprehend. In this report five devices were studied. Gibeck Humid Vent Filter, Pall HME, Portex Thermo-Vent 600, Gibeck Humid Vent 1, and Siemens Servo Humidifier 150. In this group, the size of the devices allows the three largest—Gibeck Humid Vent Filter (63 mL dead space), Pall HME (95 mL dead space), and Siemens 150 (90 mL/dead space)—to be compared. Comparing these two HCHs to the HME does in fact provide the data we suggested. Dr Sessler then states that they concluded the differences among tested units "were not clinically important." Perhaps that is true in a study in which short-term use in the operating room is required. However, evidence suggests that the low moisture output of the Pall HME is clinically important because a number of studies have shown endotracheal tube occlusions associated with its use.[3,4,6,8] Certainly the death of a patient in the study by Martin et al[4] can be considered clinically important. As further proof, however, we offer the recent paper by Sottiaux et al published in July 1993.[10] In a group of 29 surgical patients requiring mechanical ventilation, 11 received humidification with the Pall HME, 8 with the Gibeck Humid Vent, and 10 with the Hygrobac DAR. All patients were initially ventilated at a tidal volume of 10 mL/kg, respiratory rate of 14 breaths/min, and an F_{IO_2} of 0.3. Measurements of temperature and absolute humidity were accomplished at several points in the patient-ventilator system. Sottiaux et al found that the Pall HME provided less humidity than either of the two hygroscopic devices (Gibeck and DAR 28 g H_2O/m^3 vs 22.75 g H_2O/m^3 for Pall). Let Sottiaux and colleagues[10] speak to Dr Sessler's objections:

> Referring to minimal criteria published by several authors, the Pall filter appears to be unsatisfactory, even with the relatively low V_T. Several studies have corroborated the poor performance of the Pall filter. Recently a study was interrupted after the death of a patient in a BB50 Pall group from total obstruction of an ETT.
>
> The performance of the hydrophobic HME (Group 1) was inferior and appears to be unsatisfactory.
>
> Performance of the hydrophobic HME may be weak and can expose the patient to an unacceptable risk of endotracheal tube occlusion.

Again we miss the point of Dr Sessler's objections. He feels we misrepresented his data and that the differences in devices are not clinically significant. The authors of four published studies certainly disagree with this contention and the reported incidence of tracheal tube occlusion is certainly reproducible.

We made three errors in the section on the influence of inspired gas temperature on body temperature. Unfortunately, two occur in the same conversion equation. The correct conversion equation is

$$1 \text{ kcal/h} = 4.19 \text{ kJ/h} = 1.16 \text{ W}.$$

However, these errors do not affect the subsequent analysis and conclusions. The estimation of 17 W of heat energy lost from the adult expired air is taken from Walker et al[11] who state a value of 350 kcal per day. The conversion to watts is

$$\frac{350 \text{ kcal}}{\text{day}} \times \frac{1 \text{ day}}{24 \text{ h}} \times \frac{1.16 \text{ W}}{\text{kcal/h}} = 17 \text{ W}.$$

This estimation of 17 W lost through expired air is close to the 12 W estimated by Hendrickx[12] and Shanks.[13] It represents both sensible and insensible heat loss from the respiratory system only. It is also consistent with the estimate of 10-15 W quoted by Dr Sessler and his colleagues.[14] Therefore, we see no justification for Dr Sessler's remark that our estimate is "... roughly twice the correct value." There is no "correct" value. These are all 'ballpark' estimates. Dr Sessler should appreciate this as he has also estimated a respiratory heat loss of as low as 7 W in another paper,[8] which would make one of his own estimates twice the other.

According to Allen,[15] basal metabolic heat production is about 46 W/m^2. An 'average' adult weighing 68 kg and 170 cm tall (not 74 cm tall, as we erroneously stated in the text) has a body surface area of about 1.8 m^2 (from the Dubois body sur-

face nomogram). The heat production of this average adult is therefore 46 $W/m^2 \times 1.8\ m^2 = 82.8$ W. Thus, the fraction of heat lost from expired gas is 17 W/82.8 W = 0.21 (or 21% as stated in the text). We did not ". . . calculate respiration heat loss from the fraction of total heat loss which is evaporative." As Dr Sessler suggested, we calculated it from total sensible and insensible respiratory heat loss and total metabolic heat loss. We have not confused total evaporative loss with respiratory heat loss as Dr Sessler assumes, and 21% is not twice any "correct" value.

Regardless of the estimate used (and Dr Sessler's low estimate simply reinforces our point), the take-home message is still that the heat lost from the respiratory system accounts for a small fraction of the basal metabolic heat production and thus *". . . heating inspired gases contributes little to total body rewarming."*[16] As Baumgarten has pointed out, ". . . many anesthesiologists feel that heated humidifiers actively warm a patient who has been allowed to get cold.[17] The article by Ralley et al[16] dispels this myth."

Dr Sessler states that "Branson and Chatburn's unique calculations require a series of assumptions, at least some of which are incorrect." In our text we gave the exact equations required to calculate heat balance from a detailed article in the Journal of Applied Physiology.[18] We gave the explicit assumptions upon which the equations are based (ie, gas density, specific heat, and volume are assumed to be the same for inspired and expired gas). We attempted to summarize the equations with the statement that "Basically, heat exchange is proportional to minute ventilation and either temperature or water vapor content gradients," which is essentially the same as (if not more precise than) Dr Sessler's statement that "minute ventilation and inspiratory and expiratory gas temperature and relative humidity are the only information required to calculate respiratory heat balance." The calculations provided in this letter to justify the ideas in our paper[1] involve no assumptions other than those assumed by authors (ie, Walker et al[11] and Allen[15]), and estimates of heat loss are adequate for demonstration purposes.

Dr Sessler missed the point of our discussion of rewarming protocols. As we said in the text, previous studies of rewarming from accidental hypothermia or even hypothermia after anesthesia[19] have been criticized because of improper core temperature estimates[12] and a variety of other confounding factors—differences in amount of insulation provided for subjects, differences in time span between cooling and rewarming, differences in room temperature, and variations in inspired gas temperature.[20] Dr Sessler's comment about core temperature not reflecting total body heat content only emphasizes the problem with previous studies. That is why Romet and Hoskin[20] decided upon their particular study design using a *whole body calorimeter* to measure heat transfer during intentional cooling and rewarming of volunteer subjects. Body temperatures after cooling averaged 36.7°C simulating "mild hypothermia." Such body temperatures are not "irrelevant to typical clinical conditions," as Dr Sessler says and are even closer to normal than the postanesthesia body temperature reported by Stone et al.[19] Use of a calorimeter allowed these investigators to make conclusions about changes in total body heat content without having to use 'guesstimates' based on body-temperature measurements or indirect measurements of water and temperature changes as other researchers, including Dr Sessler,[8] have done. Thus, by using a calorimeter to measure body heat content directly, Romet and Hoskin[20] avoided the "...implicit assumption that changes in core temperature reasonably indicate changes in body heat content" that concerns Dr Sessler. Nevertheless, Romet and Hoskin did measure body temperature at all the major sites used by previous researchers (ie, rectum, esophagus, and auditory canal). Their results indicate that (1) esophageal temperature—the closest approximation of aortic blood or cardiac temperature—was the most sensitive to change during rewarming, (2) breathing 40°C and 45°C air accounts for only 4-7% of the total heat exchange, and (3) while breathing heated air there are reductions in metabolic rate and ventilation that are not compensated for by the small amount of respiratory heat added.

In summary, we apologize for the numerical errors, which are inexcusable. However, because Dr Sessler has provided neither a counterargument (based on either theory or experimental data) nor reference for a counterargument, we maintain our original assertion that one should not try to speed rewarming of core body temperature by increasing the inspired gas temperature above normal (ie, about 33°C).

The tone of Dr Sessler's letter disturbs us most. If his intent was to discredit us through accusations that are unsupported, he will be disappointed. We believe that readers of RESPIRATORY CARE will certainly travel to the library; retrieve, read, and digest the articles we have discussed; and come to a conclusion similar to ours. There is no evidence to suggest that use of an HME/HCH filter de-

creases the incidence of nosocomial pneumonia. Use of the Pall HME places the patient at risk for the development of tracheal tube occlusion. This isn't our opinion. It's simply a fact based on the published results of several studies.

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Mr Branson and Mr Chatburn have no financial interests in the products mentioned or in competing products.

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Reply to Paluch on HME Misrepresentations

We read with interest the letter written by Mr Paluch,[1] President of Nova-VentRX Inc, in the September issue of the Journal and would like to comment.

Heated humidifiers have been shown to provide an environment favorable for the growth of microorganisms.[2] In fact, Rhame et al[3] demonstrated that the Cascade I Humidifier produced bacteria-laden microaerosol. In addition, with a hot-water-bath system, condensation does occur in the circuitry and provides a nidus for bacterial colonization.[4] This needs to be drained and, as a result, provides the potential for further infectivity. The incredible numbers and variety of organisms that can be isolated have been well documented.[5,6] Indeed, these authors have shown that within 24 hours, 85% of

ventilator circuits, 57% of water traps, *55% of the Cascade humidifiers*, and 88% of inspiratory tubing were colonized. Also, it has been shown that medical gases are not sterile and that passage of a gas through contaminated tubing allows for the pick-up and transmission of that contamination.[7,8]

With regard to the dissemination of pathogens, Mr Paluch states that the source of the pathogen is not the reservoir—but the patient. This is generally true. However, the hot-water-bath reservoir and/or condensate becomes contaminated by retrograde microbial movement.[9,10] Once microorganisms are introduced into a warm, moist environment, rapid growth can occur. This can be clearly seen in many systems where the condensate in watertraps is turbid, indicating gross microbial contamination. In addition, Craven and his colleagues[4,11,12] caution practitioners that handling contaminated condensate poses a risk for contamination of the hands.

The Pall HME Filter *is* an effective alternative to heated humidifiers for most patients. Mr Paluch has omitted such well-respected authors as Gallagher et al,[13] Tenaillon et al,[14] Chalon et al,[15] and Bethune[16] who highlight the role of the Pall Filter in humidifying gases for ventilated patients. Indeed, Tenaillon et al[14] directly compare the Pall Filter to a Fisher-Paykel water bath and quote: "The humidifying devices were similar as regards tracheal secretions, endotracheal tube obstruction, and pulmonary complications but the filter reduced inspiratory line resistance, the costs of ventilation, and nursing time." Other papers also highlight the role of the HME/HMEF devices in long-term ventilation.[17,18] Perhaps if these references had been included, a broader perspective may have been presented in Mr Paluch's letter.

We refute that the Pall report misrepresents the performance of the Pall HMEF. It is unfortunate that Mr Paluch misunderstood our report, which says "The artificial patient temperature was maintained at 33-38° throughout the test and was monitored on an hourly basis." This refers to the range of temperature of the water bath and not temperature presented to the HMEF, as clearly illustrated in Figure 1 of our test report.[19] Our current testing apparatus is set to deliver 34 ± 1°C to the HME, as recommended by the International Standards Organization,[20] and, therefore, we feel that clinical users can judge this international performance rating. The performance of the Pall HMEF has been widely documented and the suitability and indeed the preference over a heated water bath indicated.[13,14]

Mr Paluch discussed at length the work by Cohen et al[21] and by Martin et al.[22] There is a significant omission in that there is no reference in Mr Paluch's letter to the correspondence subsequent to the Cohen paper. For a more detailed explanation, we would refer the reader to replies to Cohen by Demers[23] and Ippolito and Spallanzani.[24] With particular reference to the Martin paper the following quotation is found:

> Endotracheal toilets and instillations are performed with a strict aseptic procedure: . . .. For each patient, the frequency of tracheal aspirations or instillations is decided by a critical care physician and the nurse in charge of the patient. . . . Instillations are performed using 5 mL of sterile water once or several times in a row. *They are prescribed when pulmonary secretions became thick and tenacious.* (emphasis added).

The paper needs further explanation as it would suggest that the authors were having a "problem" with the humidification levels given by the Pall HMEF. If this was the case, the number of instillations and tracheal toilets would be expected to be very high in the Pall HMEF group, when compared to the hot water bath group. However looking at their data, we see no such differences.

	Pall HMEF	Hot Water Bath
Instillations per day	2.5 ± 0.6	2.8 ± 0.7
Bronchial toilets per day	4.0 ± 0.3	5.0 ± 0.3
But days of thick tenacious secretions	4%	0%

Indeed, 30% of patients using the Pall Filter received *no* instillation of saline at all. Obviously, the instillation and bronchial toilet frequency requires further clarification. With particular reference to Mr Paluch's comment on the death of a patient, careful reading of the paper shows there is no comment or intimation as to the role of the filter.

In terms of optimal physiologic levels of humidification, the AARC consensus statement[25] recommends 25-35 mg H_2O/L. In a recent abstract, the Pall HMEF was reported to provide > 30 mg H_2O/L.[26]

We at Pall welcome constructive criticism whether from healthcare workers or from industry. However, the criticism has to be based upon a fair representation of the available literature. In this case, we believe the criticism in Mr Paluch's letter is unjust. In addition, it would have been appropriate, as with other journals, for us to have been given the opportunity to reply to the letter in the same issue as its publication.

In conclusion, the words of Shelly a "hot water humidifier or a HME filter will satisfy the needs of most adult patients requiring artificial ventilation on an intensive care unit"[18] and "Humidification of inspired gases should not be considered in isolation but as part of total airway management. It should be associated with careful fluid balance, physio-

therapy, bronchial aspiration, and appropriate drug therapy,"[17] would seem to sum up the situation.

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Deep Breaths, Sighs, and Pulmonary Artery Catheters

I would like to pose a question for discussion among readers of the Journal—Is sighing a mechanically ventilated patient a safe and sometimes effective way to dislodge a spontaneously wedged (self-wedged) pulmonary artery catheter (PAC)?

In the intensive care unit (ICU) of our 320-bed community hospital in southern Ontario, PACs are used frequently. When the hospital's Respiratory Therapy Department was established in 1991 (as mandated by legislation) and therapists took over ventilator management in that unit, we found an established practice about which we are uncertain. The medical staff does not allow the unit's registered nurses to manipulate

the position of PACs but rather directs them to notify the most responsible physician and then position the patient to avoid complications until the patient can be assessed. However, the nurses' understanding has been that having the patient inhale deeply often may cause to the catheter to dislodge spontaneously, producing the desired results. If the patient does not respond to the nurse's request for a deep breath, logic has led them to believe that a manual sigh via the ventilator achieves similar results. According to anecdotal report, this procedure has been practised almost routinely for several years with acceptable results. However, since the Respiratory Therapy Department has assumed responsibility for ventilator management in the unit, we have requested that manual sighs not be used until the possible efficacy of this procedure can be weighed against the potential for barotrauma. It seems reasonable to assume that the mode of ventilation and the use of pressure support would affect the transthoracic pressure changes occurring during spontaneous breaths and might alter the efficacy of the manoeuvre. Conversations with a number of respirologists and therapists involved in research have yet to reveal the mode of action or potential efficacy of this procedure. In a recent literature review of the use of the sigh, I found no mention of this application. If you can shed light on the possible mechanisms involved in this procedure or expand on the potential for its use, please respond to me through the Journal.

Ted Reesor RRT
Joseph Brant Memorial Hospital
Burlington, Ontario, Canada

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Calendar of Events

AARC & AFFILIATES

October 27-28 in Sturbridge, Massachusetts. The MSRC presents its 16th Annual Meeting at the Sturbridge Host Hotel and Conference Center. Topics, include new modes of ventilation, computers in respiratory care, therapist-driven protocols, smoking cessation, neonatal flow-sync ventilation, and a debate on modes of aerosol drug delivery. AARC Executive Director Sam Giordano MBA RRT, presents the keynote address. Social events include Sputum Bowl, golf tournament, awards banquet, and dance. Contact Bill McGarry at the MSRC Executive Office, 945 Concord St., Framingham MA 01701, (508) 620-4505.

October 28-29 in Merrillville, Indiana. Chapter I of the ISRC presents the 4th Annual Harvest Seminar at the Radisson Hotel Star Plaza. Lunch, continental breakfast, and harvest hoedown dance are included with registration. For information, contact: Jo Ann Laudani, Respiratory Care Dept, St Margaret Mercy, (219) 933-2014.

November 4-6 in Hilton Head, South Carolina. Low Country AHEC and the SCSRC announce their 3rd Annual Fall Pediatric Symposium. Topics include Pediatric Emergencies, Active Cycle Breathing Techniques, and Ventilator Workshop. For details, contact David Hayden at (803) 943-5052.

November 12 in Nashville, Tennessee. The TSRC holds its Annual Meeting in conjunction with the Tennessee Hospital Association's convention and exhibition at the Opryland Hotel. Speakers/topics include Senator Robert Rochelle, "TennCare—Oversight Committee Update"; John Faulkner, president and CEO, Jesse Holman Jones Hospital, "TennCare's Impact on Hospitals"; Manny Martins, Director of Medicaid, "TennCare's Impact on Home Health and DME"; Roger Richardson RRT, "Medicare DMERC Consolidation"; and Dr Robert Tallon, "New DMERC Medical Policies." For information regarding registration and/or exhibits, contact Carol McCabe at (615) 256-8240.

December 2. AARC Videoconference. The AARC, in conjunction with VHA Satellite Network, presents the sixth of a six-part videoconference series titled "Professor's Rounds in Respiratory Care." The final presentation, "Unconventional Methods for Adult Oxygenation and Ventilation Support," features James K Stoller MD, and David J Pierson MD. For information, call (214) 830-0061.

December 10 in Nashville, Tennessee. The AARC presents a postgraduate course for medical directors of respiratory care and pulmonary physicians: Part 1 —Mechanical Ventilation and Acute Respiratory Failure; Part 2 — Medical Director's Role in Respiratory Care. Designated for 6.0 credit hours in Category 1 of the Physician's Recognition Award of the American Medical Association. Tuition is $175. A continuing education program of Vanderbilt University Medical Center and the AARC. Contact Robert Czachowski PhD, Director of Education, AARC, 11030 Ables Ln, Dallas TX 75229-4593, (214) 243-2272, fax (214) 484-2720.

December 11-14 in Nashville, Tennessee. The AARC presents its 39th Annual Convention and Exhibition, "New Rules, New Roles, New Responsibilities," at the Opryland Hotel and Convention Center. For information, contact the AARC at (214) 243-2272, fax (214) 484-2720.

OTHER

October 13 in Atlanta, Georgia. St Joseph's Hospital of Atlanta presents its 10th Annual Respiratory Care Seminar. Contact: Rena Cobb RRT RCP, Education Coordinator, Respiratory Care, St Joseph's Hospital of Atlanta, 5665 Peachtree Dunwoody Rd, Atlanta GA 30342, or call (404) 851-7176.

October 14-15 in Asheville, North Carolina. The 5th Annual Mountain Air Conference for Respiratory Care features Dr Larry Pepper, manager of Shuttle Medical Operations for NASA, discussing innovations in health care delivery. Other topics include multidrug-resistant TB, hyperbaric therapy, stabilization of burn patients, and a pediatric asthma update. For more information, contact Charolette Artis at (704) 257-4473.

October 19-20 in Birmingham, Alabama. Respiratory Care Services and the Department of Neonatal Nursing of The Children's Hospital of Alabama hosts the 3rd Annual Perinatal-Pediatric Clini-Course at the Sheraton Civic Center-Medical Forum. Topics covered include Neonatal Mechanical Ventilation Algorithms, Developing Interventions in the NICU, Nitric Oxide Therapy, Customizing Negative Pressure Ventilation, Overview of Genetics, and many more. If you have any questions regarding this Clini-Course, contact: Susan Forrest, The Children's Hospital, 1600 7th Ave South, Birmingham, AL 35233, (205) 939-9675.

October 29 in Reno, Nevada. The American Lung Association of Nevada presents its 12th Annual Respiratory Health Conference in the Mack Auditorium at Washoe Medical Center. Contact: Sarah Kentfield, American Lung Association of Nevada, PO Box 7056, Reno NV 89510-7056, or call (702) 829-5864.

January 27-29, 1994 at Helena, Montana. 13th Annual Big Sky Pulmonary Ski Conference. Contact: The American Lung Association of Montana, 825 Helena Ave, Helena MT 59601, (406) 442-6556, fax (406) 442-2346.

April 2-8, 1994 in Miami, Florida. The 1994 session of Miami Children's Hospital's Annual VACC Camp for ventilation-assisted children and their families is held. Application due date is Jan. 15, 1994. This community-based, free camp program provides recreation and social support for children with trachs, vents, CPAP, etc, and their moms, dads, and siblings at least 5 years old. Technology-dependent children should have normal cognitive potential. For information, contact: Director Moises Simpser MD, or Program Coordinator Cathy Klein, Ventilation Assisted Children's Center, Division of Pulmonology, Miami Children's Hospital, 3200 SW 60th Court, Suite 203, Miami FL 33155-4076, (305) 662-VACC.

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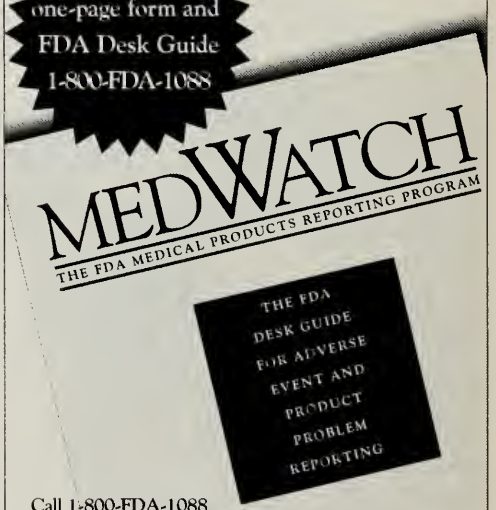
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The American Respiratory Care Foundation Awards for 1993

1. Dr Allen DeVilbiss Literary Award for the best paper published from November 1992 through October 1993 that addresses new technology or a new application of current technology in respiratory care: $2,000 cash plus travel expenses to the AARC Annual Meeting to receive the award.
2. $2,000 for the best original paper (study, evaluation, or case report) *accepted* for publication from December 1992 through October 1993. This award is *not* limited to papers based on OPEN FORUM presentations.
3. Four awards of $1,000 each for papers *accepted* for publication from November 1992 through October 1993 based on *any* OPEN FORUM presentation (not limited to 1992 OPEN FORUM).
4. Five awards of $500 each for the best papers *submitted* (not necessarily published) by 1993 OPEN FORUM participants who have 'never published' in the Journal. The never-published first author must present the abstract at the Annual Meeting and must submit a paper based on the abstract before the 1993 Annual Meeting (*received* in the Editorial Office by November 1, 1993). Co-authors may have previously published in RESPIRATORY CARE.

Three awards of $333 each are to be awarded to the authors of the three best features from Test Your Radiologic Skill, Blood Gas Corner, Kittredge's Corner, and PFT Corner accepted for publication from November 1992 through October 1993. All three (or none) of the features may be chosen from a specific category (eg, all three may be chosen from Blood Gas Corner).

All awards will be made at the 1993 Annual Meeting. Papers are judged automatically. No application is necessary.

Annual Meeting Registration Reimbursement

As in the past, any 1993 OPEN FORUM presenter (or co-author designee) will receive complimentary registration for an adequately prepared paper based on his 1993 OPEN FORUM abstract, submitted prior to or at the 1993 Annual Meeting.

THE NATIONAL BOARD FOR RESPIRATORY CARE—1993 Examination and Fee Schedule

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Perinatal/Pediatric Specialty Examination

EXAMINATION DATE:	MARCH 12, 1994
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Application Deadline:	November 1, 1993

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Akingbola, Olugbenga A 1092
Albright, Douglas C 1069
Ballard, Julie 1072
Burton, Karen 1072
Branson, Richard D 1114
Chatburn, Robert L 1114
Custer, Joseph R 1092
Deutsch Jr, E Thomas 1069
Fluck Jr, Robert R 1112
Graf, Kenneth 1117
Indihar, Frank 1081
Jaidev, Vasudevan C 1110
Kacmarek, Robert M 1103
Kennan, Jim 1072
Kennedy, Dianne L 1077
Lloyd, Geoff 1117
Lynch, Joanna 1072
Meeker, David P 1110
O'Connor, James M 1088
Ollivier, Joseph E 1107
Olson, Suzanne 1081
Palmisano, John M 1092
Reesor, Ted 1119
Robbins, Yvonne Jo 1069
Salyer, John W 1072
Servant, Gina M 1092
Sessler, Daniel I 1113
Stelmach, Kay 1110
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